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INHERITED CATARACT IN THE B. GENEALOGY

OCCURRENCE OF DIVERSE TYPES OF CATARACT IN THE DESCENDANTS OF ONE PERSON

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The numerous publications on developmental cataract, as summarized by Nettleship,[1] Harman,[2] Groenouw,[3] Clausen,[4] and Waardenburg,[5] have tended to show a uniformity in the type of cataract observed within any family line. Nevertheless, it is sometimes found that certain of the cataractous persons in a given pedigree have a type of opacity of the lens significantly different from that presented by the remaining affected members of the family (Knies,[6] Gjersing, 1878, quoted from Harman,[2] Nettleship,[7] Harman,[8] Groenouw,[3] Halbertsma,[9] Walsh and Wegman[10]). This fact has been commented on by various observers, and a question raised from time to time as to the etiologic relationships of the various types. The present paper will describe a genealogy of 123 people, 44 with cataract, which is remarkable for the variety of forms the cataract assumes, and will discuss the question of the genetic relationship of the various types of cataract seen in this and comparable pedigrees.

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1. Nettleship, E.: On Heredity in the Various Forms of Cataract, Roy. London Ophth. Hosp. Rep. **16**:179, 1905; **16**:389, 1906.

2. Harman, N. B.: Congenital Cataract, in Treasury of Human Inheritance, edited by K. Pearson, London, Cambridge University Press, 1912, vol. 1, p. 126.

3. Groenouw, A.: Beziehungen der Allgemeinleiden und Organerkrankungen zu Veränderungen und Krankheiten des Sehorgans, in Graefe, A., and Saemisch, T.: Handbuch der gesamten Augenheilkunde, ed. 3, Leipzig, W. Engelmann, 1920.

4. Clausen, W.: Vererbungslehre und Augenheilkunde, Zentralbl. f. d. ges. Ophth. **11**:209, 1924.

5. Waardenburg, P. J.: Das menschliche Auge und seine Erbanlagen, The Hague, Martinius Nijhoff, 1932.

6. Knies, M.: Ueber den Spindel-Staar und die Accommodation bei demselben, Arch. f. Ophth. **23**:211, 1877.

7. (*a*) Nettleship, E., and Ogilvie, M.: A Peculiar Form of Hereditary Congenital Cataract, Tr. Ophth. Soc. U. Kingdom **26**:191, 1906. (*b*) Nettleship, E.: Lamellar Cataract, "Coppock" or Discoid Cataract, and Retinitis Pigmentosa, Affecting Different Members of the Same Pedigree, ibid. **28**:226, 1908.

8. Harman, N. B.: Congenital Cataract: A Pedigree of Five Generations, Tr. Ophth. Soc. U. Kingdom **29**:101, 1909.

9. Halbertsma, K. T. A.: Ueber familiäre juvenile Katarakt, Klin. Monatsbl. f. Augenh. **80**:108, 1928.

10. Walsh, F. B., and Wegman, M. E.: Pedigree of Hereditary Cataract, Illustrating Sex-Limited Type, Bull. Johns Hopkins Hosp. **61**:125, 1937.

THE B. FAMILY

The persons comprising this genealogy are the descendants of one William B. (I 1) who, in 1849, at the age of 24, migrated from Chelmsford, Essex County, England, to Ontario Center, in the northwestern part of the state of New York, when this region was first settled. He was known to have cataracts, and with his poor vision was able to do only farm and outdoor labor. He could never see well enough to read, and in his later years his vision became progressively worse until he was unable to get around outside of his house. He died at the age of 74 of cancer. W. B. was believed to have inherited the cataracts from his mother. Nothing further back is known of the family, except that there were other members who remained in England who were also afflicted with cataracts. No other members of this family are known to have migrated to America. Two of B.s' daughters (II 6 and II 10), who are still living, supplied the foregoing information and contributed greatly to the construction of the pedigree (fig. 1).

The first members of the family whom we saw were IV 46 and IV 47, when they came to the Eye Clinic of the Strong Memorial and Rochester Municipal Hospitals. IV 13 was seen next, and the various members of his line of the family were then checked. This was followed by an examination of II 10 and her descendants. Other members of the family were examined as the opportunity presented itself. Additional information was gathered about the family from almost all of those seen. Most of the members of the family have remained in the same section of the country, living within a few miles of each other, and a fairly close family contact has been maintained, so that it is well known within the family which members have cataract, except for

the youngest generation. Confirmation of the presence of cataract was usually made by more than one person in those cases in which we were unable to make an examination. Newborn infants in the family were watched carefully by the parents and the relatives for the first evidence of visual difficulty, and those having cataract were known from early childhood to have subnormal vision. In no member of the family who was unaffected with cataract in childhood was cataract known to develop later in life.

The majority of the family, whose industry is average, have remained in rural districts engaged in work such as that required of laborers, farmers and unskilled workmen. A few have been factory workers. No one in the family either with or without cataract has achieved unusual prominence or success in any field of work; nor were any persons encountered of the economically dependent and shiftless type. The homes that were visited were average for their economic level. Evidences of physical, moral or mental degeneracy in the family were absent. Two persons, III 15 and IV 28, were said to be of subnormal mentality. However, all whom we examined were of good physique and skeletal structure, with no evidence of rickets, and of apparently average intelligence. Some of the children, both with and without cataract, were of unusually attractive appearance. The teeth of nearly all the cataractous persons examined were inspected; there were no greater than average incidence of caries, and no malformations. Only one person, IV 47, had a few horizontal ridges on his teeth. No history of convulsions during infancy was elicited from any member of the family.

Most of those with cataract whom we saw had had only one eye treated surgically, and with this eye possessed approximately normal corrected vision. In a majority of the cases the surgical treatment up to about the age of 30 had been repeated discissions. The cataract was unaccompanied by any other structural abnormalities of the eye. A complicated myopia occurring in III 18 and some of her descendants required thin cataract lenses to correct the eyes after operation. III 18 had a satisfactory intracapsular lens extraction, but high myopic degeneration caused poor vision. IV 13, who also had extensive myopic retinal degeneration, had had bilateral extracapsular lens extractions followed later by retinal separations. This type of myopia did not occur in the other lines with the cataract. In only one instance (V 5) had surgical treatment consisting of discission of one eye been followed by complications which were disastrous to vision.

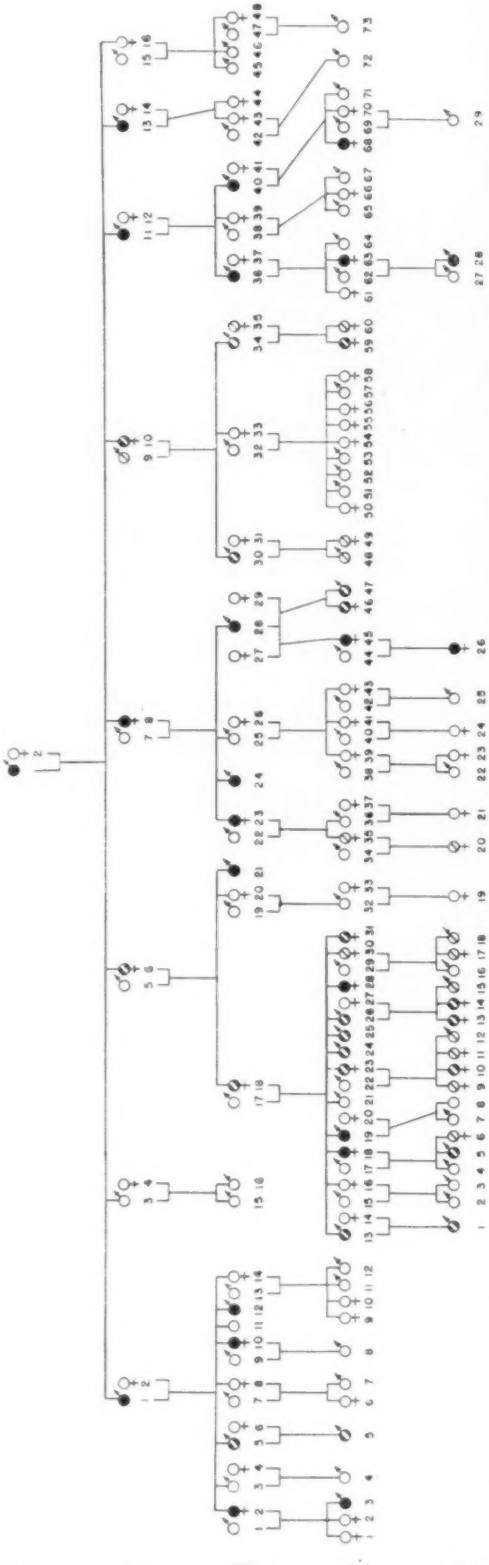

Fig. 1.—The pedigree of the B family. The circle with the diagonal line through it indicates normal eyes, examined; the white circle, eyes reported normal but not examined; the circle with the heavy black border, eyes with cataract, examined; the black circle, eyes reported cataractous, not examined.

A detailed description of the B. genealogy follows:

II 1: D. B. had cataracts. His vision had always been poor. He was never able to read newsprint even between his fingers. His vision gradually became worse, so that one cataract was removed at the age of 60. He died at the age of 74 years.

II 3: G. B. had good vision throughout his life, and never wore glasses. He died at the age of 74 years of heart disease.

II 6 (fig. 2): E. B. B., aged 82, had cataracts. As a child at school she had difficulty with her vision, which during later years gradually became worse. The cataract in the right eye was removed at the age of 60, with good corrected vision following. Ophthalmoscopic examination of the left eye showed a complete white lens opacity.

II 8: S. B. C. had cataracts. She had poor vision throughout life, becoming worse with age. Operation was not performed. She died at the age of 77 after prolonged heart trouble.

II 9: Ophthalmoscopic examination was performed. The lenses were clear.

II 10 (fig. 3): R. B. D., aged 74, had cataracts. She had always had difficulty reading and was able only to read newsprint between her fingers with her hand shading her eyes, or when in a dark corner of a room. She was never able to see well in a bright light. On ophthalmoscopic examination through pupils dilated to 8 mm., a ring was seen encircling the middle half of the lens. In the axial region were radiating, linear opacities, mixed with flakes and vacuoles, the latter being more common in the anterior layers. In the anterior layers of the peripheral third of the lens were radiating opacities and vacuoles.

II 11: C. B. had cataracts. His vision had always been poor, but he was able to see better at night than in bright sunlight. Both cataracts were removed at about the age of 65. He died at the age of 80.

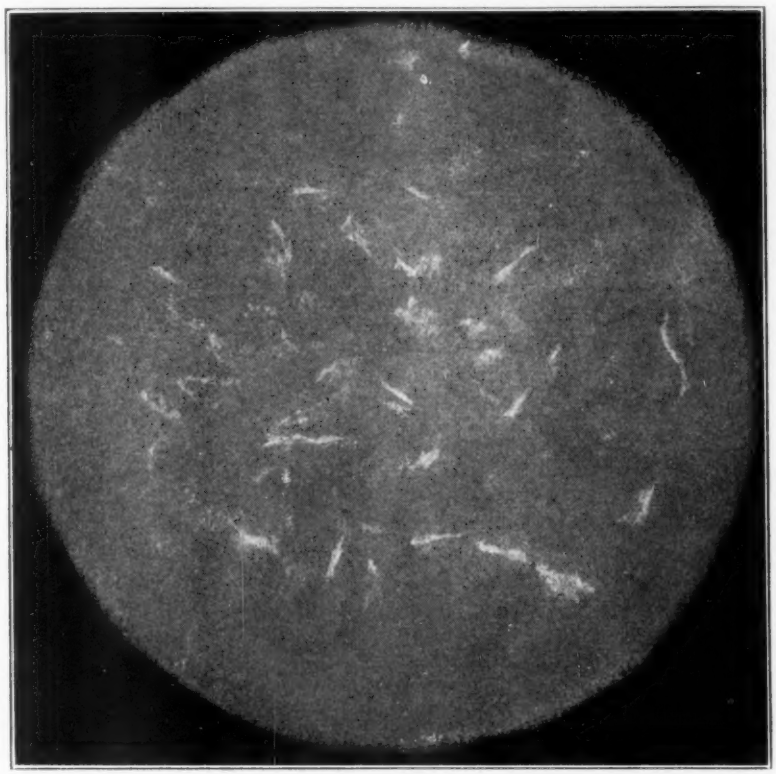

Fig. 2.—Ophthalmoscopic view of the cataract in II 6 (E. B. B., aged 82).

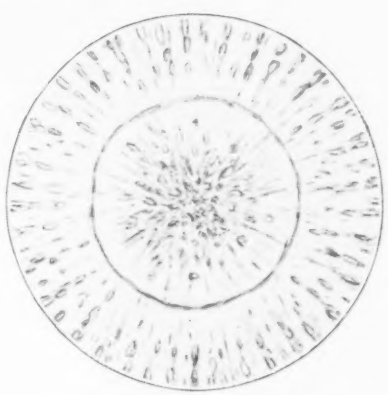

Fig. 3.—Ophthalmoscopic view of the cataract in II 10 (R. B. D., aged 74).

II 13: W. B. had cataracts. He had poor vision throughout his life, and was forced to do outdoor work. He died at the age of 55 of a sudden heart attack.

II 16: M. B. R. had good vision and could sew and read newsprint. Death occurred at the age of 69.

III 2: G. B. McF., aged 41, had cataracts. One eye was operated on.

III 3: C. B. had good eyesight. Death occurred at the age of 20 from accidental scalding.

Fig. 1.—The pedigree of the B family. The circle with the diagonal line through it indicates normal eyes, examined; the white circle, eyes reported normal but not examined; the circle with the heavy black border, eyes with cataract, examined; the black circle, eyes reported cataractous, not examined.

III 5: D. B. Jr., aged 35, had cataracts. Both cataracts were needled.

III 8: G. B. V., aged 32, had good eyesight.

III 10: E. B. M., aged 51, had cataracts.

III 11: This child died at 3 years of age.

III 12: L. B. had cataracts. He had extremely poor eyesight. He died at 35 years of age of tuberculosis.

III 21: M. B., aged 56, a storekeeper, had cataracts. One eye was operated on at the age of 36. Vision in that eye with glasses was good.

III 23: J. C. M., aged 70, had cataracts. All her life she was forced to hold things very close. She sewed and read with difficulty. One eye had been operated on; the resulting vision was unknown.

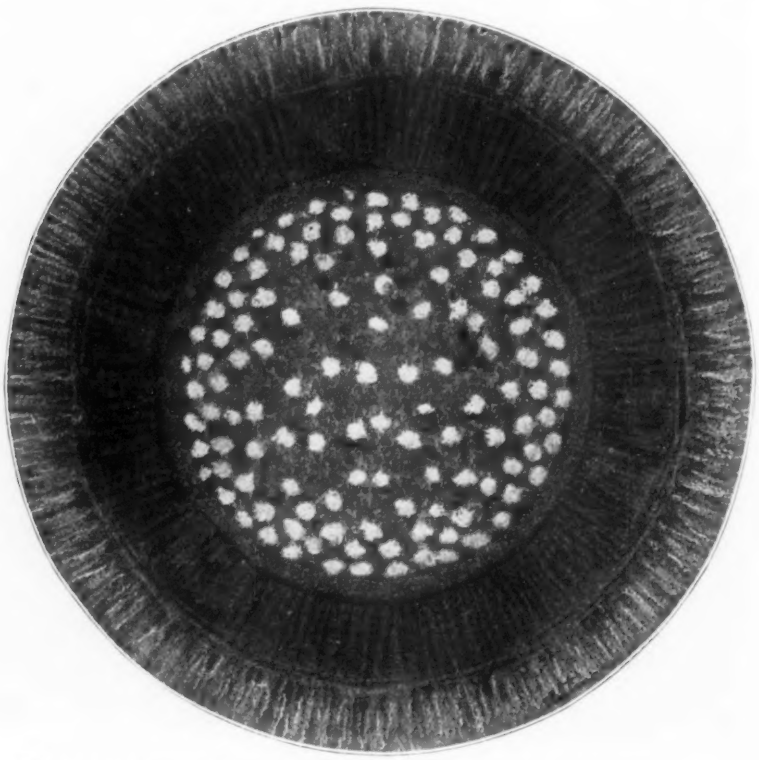

Fig. 4.—Slit lamp view of the cataract in III 18 (M. B. H., aged 61).

III 14: C. B. B., aged 44, had good eyesight.

III 15: Eyesight was good. He was described as feeble-minded. Death occurred at the age of 48.

III 16: A. B., aged 50, had good eyesight.

III 18 (fig. 4): M. B. H., aged 61, had cataracts. Vision had always been impaired. She was unable to go far in school because of her eyesight. In 1938, intracapsular cataract extraction with iridectomy was performed on her right eye. Retinoscopic examination revealed vision in the right eye with a + 6.50 D. sph. ⌒ + 2.50 D. cyl. to be 13/200. The media of this eye were clear. There was a myopic conus with retinal degeneration, extending 4 disk diameters temporal to the disk. On the lower and temporal peripheries were areas of retinal degeneration. Vision of the left eye equaled hand movements. Through the dilated pupil there was a dull red reflex around a central sphere, which was diffusely opaque, and in which were large whitish bodies scattered through all depths. Each of these bodies was a uniformly white spherical opacity with slightly blurred borders. Surrounding the large sphere was a thin zone of fine punctate opacities and farther out a linear zone. In the peripheral outer fourth in the deeper layers of the anterior cortex was a fringe of radiating opaque spokes. The posterior layers of the lens contained radial opacities.

III 20: M. B. B., aged 51, had no difficulty with vision.

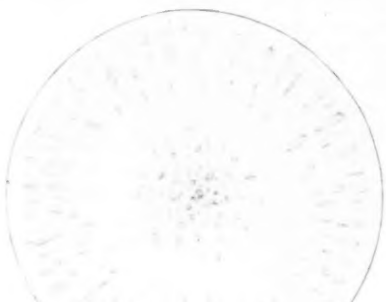

Fig. 5.—Ophthalmoscopic view of the cataract in III 30 (R. D., aged 48).

III 24: B. C., aged 61, had cataracts. He always had extremely poor vision and stumbled over things until one eye was operated on, at about the age of 36.

III 25: A. C., aged 60, had good vision.

III 28: W. C. had cataracts. He preferred a dim light, in which he could read larger newsprint type; he was unable to read in bright sunlight. He died at the age of 52 of a sudden attack of heart disease.

III 30 (fig. 5): R. D., aged 48, had cataracts. He always had had some difficulty in reading. His vision had gradually become poorer. An ophthalmoscopic examination through pupils dilated to 8 mm. revealed around the middle third of the axis of the lens, and located more in the anterior layers, scattered, fine opacities together with numerous larger round ones which appeared as vacuoles. In the outer third of the lens were radiating clefts and vacuoles. The extreme periphery of the lenses was clear. The fundi were normal.

III 33: E. D. F., aged 41, had no visual difficulty.

III 34 (fig. 6): G. D., aged 36, had cataracts. He reads Ishihara color perception charts readily. Paredrine hydrobromide [11]–homatropine hydrobromide was used as a cycloplegic for refraction:

Vision with a + 1.75 D. cyl., ax. 70 in the right eye was 6/10-2; in the left eye with a + 1.75 D. cyl., ax. 85 it was 6/10-1.

In the deeper zones of the lens in the axial region were many powdery, much finer clumps of crystals, of brilliantly reflecting colors. In addition there were occasional small flakes. A few fine granular opacities were in the posterior zones. The fundi were normal.

III 35: Paredrine hydrobromide–homatropine hydrobromide was used as a cycloplegic for the refraction:

Vision in the right eye with a — 1.75 D. sph. ⌒ — 1.00 D. cyl., ax. 55 was 6/5-3; in the left eye with a — 1.75 D. sph. ⌒ — 0.75 D. cyl., ax. 80 it was 6/5-4. Media, disks and macula were normal.

III 36: F. B., aged 69 years, had cataracts.

III 39: M. B. S., aged 60 years, had good eyesight.

III 40: W. B. had cataracts. Death occurred at 62 from cancer.

III 43: E. B. J., aged 48, had good vision.

III 44: H. B. D., aged 45, had good vision.

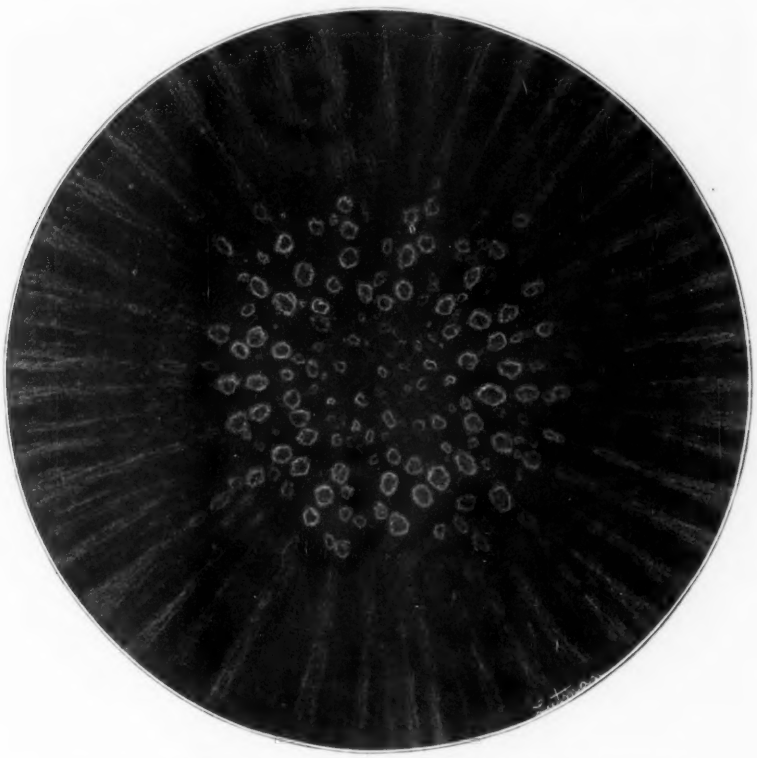

Fig. 6.—Slit lamp view of cataract in III 34 (G. D., aged 36).

By retinoscopic examination, opacities were seen on the axial half of the lens without a sharply limited zonular distribution. With the slit lamp the opacities were seen to be larger and less numerous than those in his daughter IV 59 (fig. 11), and were located in the anterior subcortex and in the anterior adult nuclear region. In the periphery of the deep anterior cortex there was a radial ring composed of spear-shaped opacities and clefts.

By direct illumination, the individual opacities resembled flakes of mica; the central portion of each was nearly clear, with its outer ring extremely iridescent.

11. The composition of paredrine hydrobromide is parahydroxy-α-methylphenylethylamine hydrobromide, 1 per cent, made isotonic with sodium chloride and preserved with merthiolate, 1: 100,000.

III 45: M. R. had good vision. Death occurred at 61 from cancer.

III 46: C. R., aged 62, had good eyesight.

III 48: P. R. S., aged 60, had good vision.

IV 1, M. McF., aged 17, and IV 2, B. McF., aged 13, both had good eyesight.

IV 3: G. McF., aged 10, had cataracts and eyesight was poor.

IV 4: G. B., aged 21, in the army, had good eyesight.

IV 5: This member of the family, aged 5, had cataracts. Both lenses had been needled once.

IV 6, S. V., aged 11, and IV 7, aged 6, had good eyesight.

IV 8: L. M., aged 26, did not have cataracts.

IV 9, aged 24; IV 10, G. B., aged 21; IV 11, F. B., aged 19, in the army; and IV 12, W. B., aged 8, all had good eyesight.

IV 13: E. H., aged 43, had cataracts. Both cataracts were removed with iridectomies. Retinal separation in the right eye occurred in 1936. For a retinal separation in the left eye in 1943, operation was performed with perforating scleral diathermy, with successful restoration of reading vision. Although the fundi could not be seen clearly, extensive myopic degeneration was visible.

IV 16: G. H. J., aged 41, had no trouble with vision.

IV 18 (fig. 7): O. H. R., aged 41, had cataracts. He worked as a factory inspector. He had great difficulty at school. The cataract in the left eye was needled four times when he was 17 years of age. The left pupil was clear. Numerous vitreous opacities were seen. A 0.5 disk diameter white conus was seen below the left disk. The fundus was normal. Vision in the left eye with a + 6.00 D sph. ⁻ + 1.50 cyl., ax. 60 was 6/7.5; + 3.00 D. sph. was added to read Jaeger's test type 2 at 36 cm. Vision in the right eye was limited to hand movements. The central half of the lens was visible through a dilated pupil as a slightly irregular spherical opacity with a narrow zone of very fine white

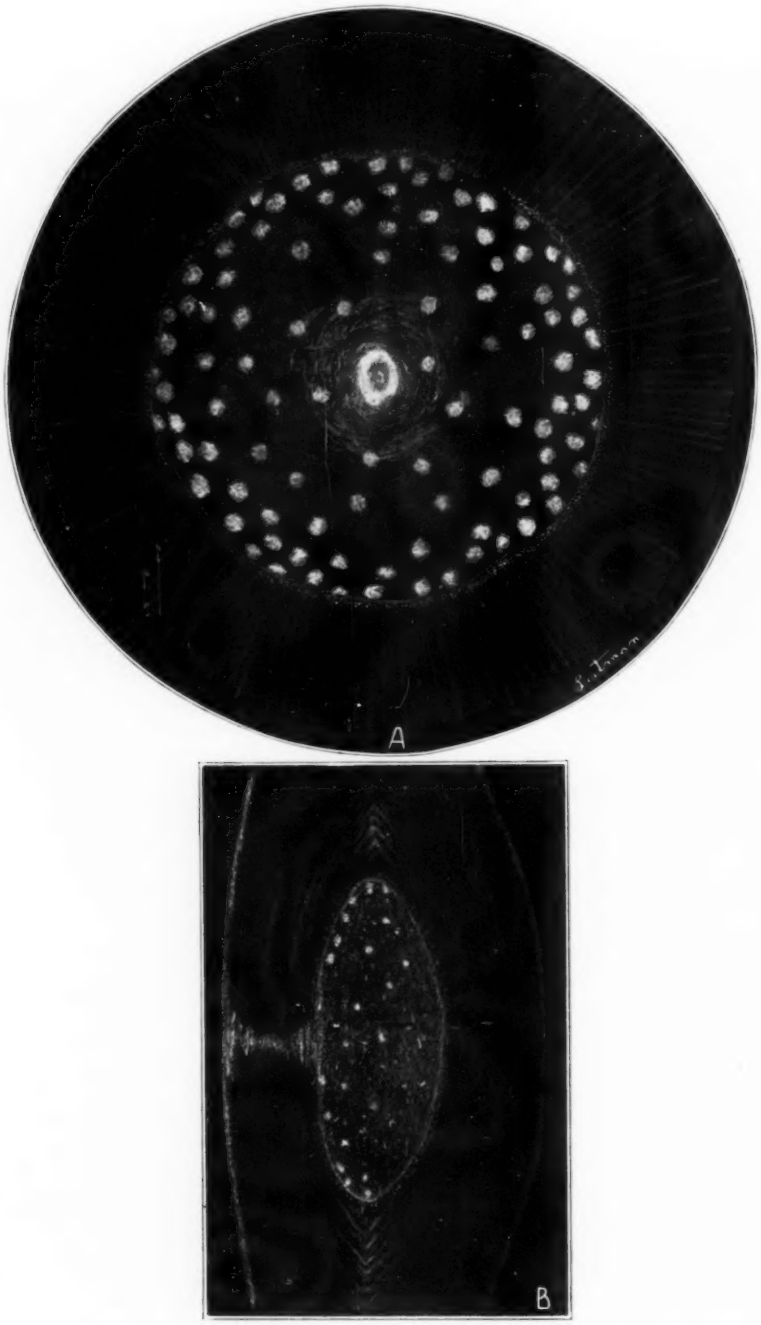

Fig. 7.—*A*, slit lamp view of the cataract in IV 18 (O. H. R., aged 41); *B*, schematic cross section of the lens in the same person.

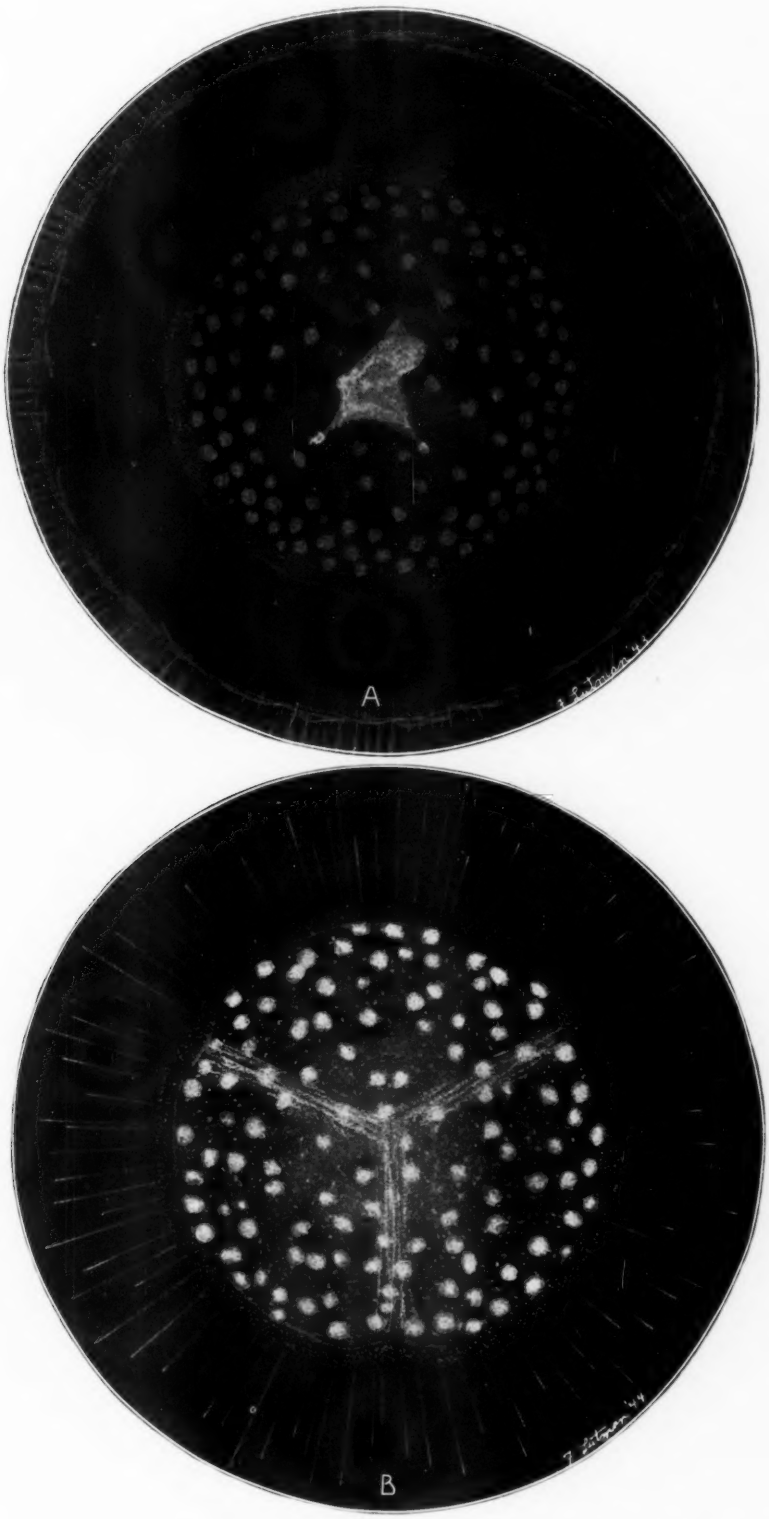

Fig. 8.—*A*, slit lamp view of the cataract in IV 23 (M. H. D., aged 33). *B*, slit lamp view of the cataract in IV 24 (A. H., aged 34).

specks surrounding it. The central nuclear opacity had flakelike opacities scattered throughout, more densely in the periphery, together with many finer punctate ones. These larger flakes possessed some thickness and a uniform opaqueness. An oval anterior polar opacity continued backward along the lens axis as a fusiform opacity to the nucleus. Opaque spokes were visible at different depths peripheral to the fetal nucleus, forming zonular riders.

IV 19: M. H., aged 37, had cataracts. One eye was believed to have been operated on.

IV 21: S. H., aged 35, had good vision with the aid of glasses.

IV 23 (fig. 8 *A*): M. H. D., aged 33, had cataracts. The right eye was needled at the age of 16. Vision in this eye with correction was 6/6; in the left eye it was 2/200. Through the dilated left pupil the central half of the lens was a sharply outlined semiopaque sphere with opaque flakes at all depths, a greater number toward the periphery of this area. The individual flakes were uniformly dull opacities with slightly blurred margins, and were scattered through all depths of the nucleus, though less numerous in the deeper layers. The central two thirds of this nucleus was a dull opaque region rather clearly demarcated. A faint ring of powdery white spots sharply outlined the fetal nucleus. A thin line circled a more peripheral zone. Close to the periphery of the lens was a ring of slightly yellowish flakes; extending from the periphery in the anterior part of the cortex and limited to a narrow zone were radial, linear opacities which passed toward the center. At the anterior pole of the lens was a triangular iridescent white opacity with an opacity continuing backward to the nucleus.

IV 24 (fig. 8 *B*): A. H., aged 34, had cataracts. He worked as a factory packer. He attributed his inability to go further than eighth grade in school to his poor eyesight. The cataract in his right eye was needled three times at the age of 21. The left eye was divergent 15 degrees. Vision in the right eye with a +13.00 D. sph. ⌒ +2.00 cyl., ax. 20 was 6/6. The right disk showed slight physiologic cupping. The retina and macula were normal. Vision in the left eye was 3/200. With a retinoscope, through the dilated left pupil, the central half of the lens was seen to be occupied by a sharply delineated opacity without a red reflex; a red reflex existed peripheral to this. With the slit lamp the opaque, anterior Y suture of the fetal nucleus was clearly visible on the anterior surface of this central opaque sphere. The three arms formed notches at the periphery of the nucleus, each notch marked by an indentation of the thin zone of fine white opacities immediately surrounding the nucleus. Solid, grayish, oval and spherical bodies, somewhat denser toward the peripheral zone, were scattered through the anterior part of the fetal nucleus when examined by section. The central portion of the lens was a milky opaque zone. Peripheral to the fetal nucleus were fine white lines which, when followed individually through the different depths, were anterior and posterior fibers joining in an acute angle toward the periphery to form riders.

IV 25: R. H., aged 33, had cataracts. He worked as an elevator operator. The cataract in the right eye was needled when he was 20 years old. He had always had poor vision, and completed the eighth grade at school with difficulty. Exotropia of 12 degrees was present in the left eye. Vision in the right eye with a +9.50 D. sph. was —6/7.5-1. Slight physiologic cupping of the optic disk was seen. The retina was normal. Vision in the left eye was 2/200. The central half of the lens was an opaque sphere surrounded by a narrow zone of very fine dots, which in turn was surrounded by a second narrow peripheral zone that was faintly opaque. In the fetal nucleus were solid, white, round and oval bodies scattered at all depths, more of them anteriorly, where the spheres were arranged somewhat radially. Others were toward the periphery of the nucleus. An anterior fusiform opacity continued from the fetal nucleus to the anterior capsule of the lens where, at the anterior lens pole, there was a small, shining capsular opacity. Peripheral to the fetal nucleus were linear, radiating white riders.

IV 26 (fig. 9): M. H., aged 30, had cataracts. He worked as a farmer. The left eye had been needled several times in the past year. An opacity still remained in the pupillary region. Vision in the right eye was 6/20-1. Through a dilated right pupil a red reflex was present which was broken up by numerous opacities radiating from the densest portion along the axis of the lens. Slit lamp examination showed a feather-like arrangement of opaque lens fibers along the sutures of the adult nucleus. Three small capsular and subcapsular opacities existed at the anterior pole. In the lower temporal periphery there was a single group of radiating opacities in the cortex. In the posterior subcortical and nuclear regions were similar opaque lens fibers along the sutures. In the cross section there was a zone deeper in the adult nucleus composed of very fine opaque strands. More centrally was a zone of faint minute specks, surrounding a central hazy opacity, in the center of which was an irregular dense white opacity. There were no flakelike opacities visible in the lens.

IV 28: D. H., aged 28, had cataracts. This person was seen but not examined. She was of subnormal mentality.

IV 30: E. H. C., aged 26, had clear lenses as revealed by examination with a retinoscope through dilated pupils. With the slit lamp, approximately fifteen fine flakelike opacities were seen in the right eye posterior to the anterior Y suture, and on the left lens, in a similar position, about ten.

IV 31: B. H., aged 23, had cataracts. The cataract in the right eye had been needled, and vision in this eye was 6/6 with a lens. Through the dilated left pupil an opacity of the fetal nucleus was observed with a narrow zone of fine powdery opacities surrounding it. In the fetal nucleus were scattered flakes, with a greater number toward the periphery, each flake a grayish opacity with a white center. Peripheral to the fetal nucleus were radiating anterior and posterior opaque fibers forming riders, and near the extreme periphery a ring of large white flat flakes.

IV 32: V. B., aged 30, had good vision.

IV 35: B. C. D., aged 40, had good vision with glasses. Slit lamp examination of the lenses showed only an occasional punctate opacity, no more than average.

IV 36: E. M., aged 38, had good vision. He wore no glasses.

IV 39: H. C., aged 35; IV 41, F. C., aged 33; and IV 43, M. C., aged 30, all had good eyesight.

IV 45: B. C. S., aged 36, had cataracts. The left lens had been needled, with a good result. The family described the right eye as having a very noticeable white opacity in the pupil.

IV 46: D. C., aged 16, had cataracts. In 1937, a sketch of her lenses as recorded on her chart showed a nuclear opacity such as occurred in most of the descendants of III 18. Refraction, with homatropine hydrobromide used as a cycloplegic, showed vision in the right eye with a —0.75 sph. ⌒ —1.50 cyl., ax. 180

to be 6/20 + 1; in the left eye, with —0.50 sph. ⌒ —1.25 cyl., ax. 170, 6/15 + 2. The cataract in the right eye was needled twice in 1937, that in the left eye twice in 1938. In 1940, an alternating exotropia developed. Refraction in 1943 showed vision in the right eye with + 12.00 sph. ⌒ + 1.50 cyl., ax. 94 to be 6/6-2; in the left eye with a + 12.00 sph. ⌒ + 1.25 cyl., ax. 90, 6/5. There was a 15 degree right exotropia. On the right after-cataract were three small, spheroid white bodies similar to those found in IV 47 (fig. 10). The fundi were normal. Ishihara charts were rapidly read.

IV 47 (fig. 10): R. C., aged 12, had cataracts. The left lens was needled twice in 1940. The right lens was needled in 1941, and again in 1943 for a wrinkled posterior capsule. As first needling in each eye had been done before the patient came under our observation, the type of cataract is unknown. In three clusters on the posterior surface of the right cornea were 26 translucent, grayish, egglike globules an estimated 0.1 to 0.2 mm. in diameter. An additional 33 of these bodies were scattered over the lower nasal iris. Several of the bodies were found in the lower left iris. Vision in the

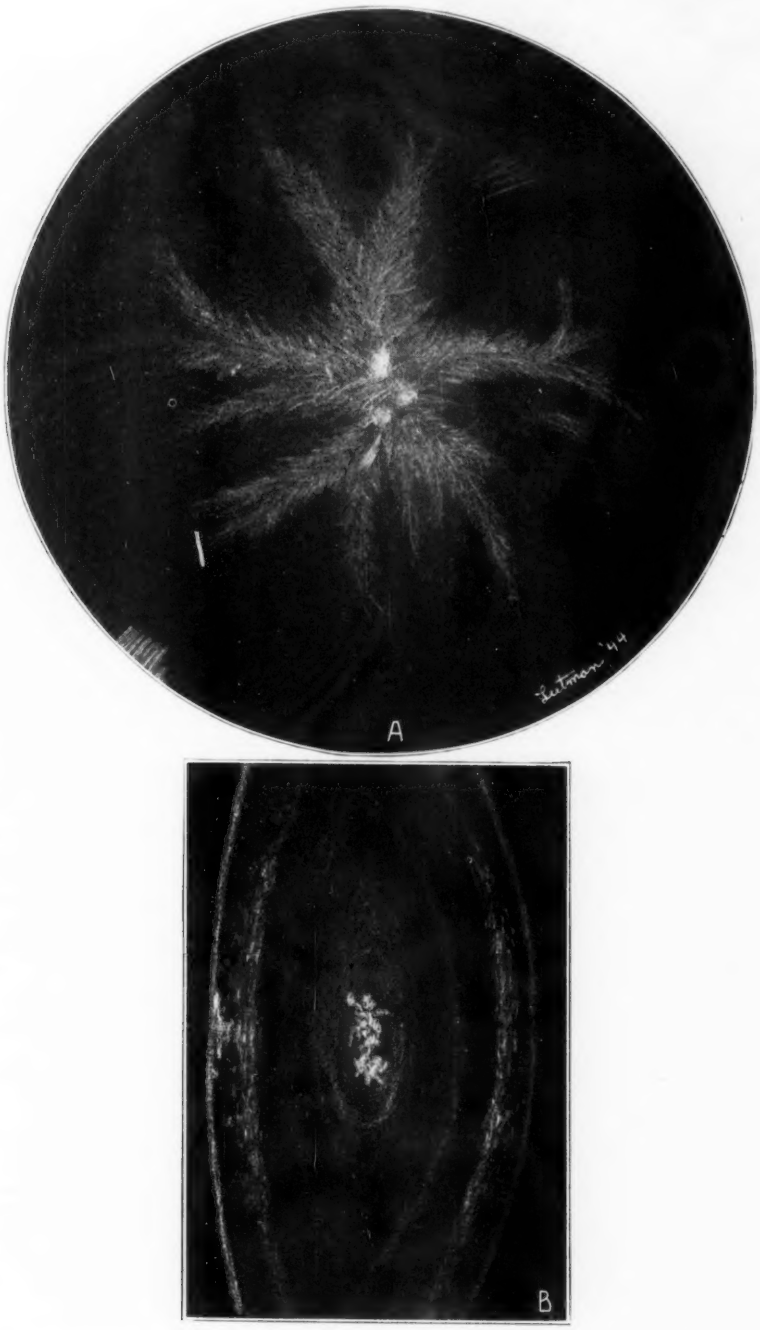

Fig. 9.—*A*, slit lamp view of the cataract in IV 26 (M. H., aged 50). *B*, schematic cross section of the lens in the same person.

right eye with a + 12.75 D. sph. ⌒ + 1.00 D. cyl., ax. 90 was 6/7.5 + 2; in the left eye with + 12.50 D. sph. ⌒ + 1.75 D. cyl., ax. 90, 6/7.5 — 2.

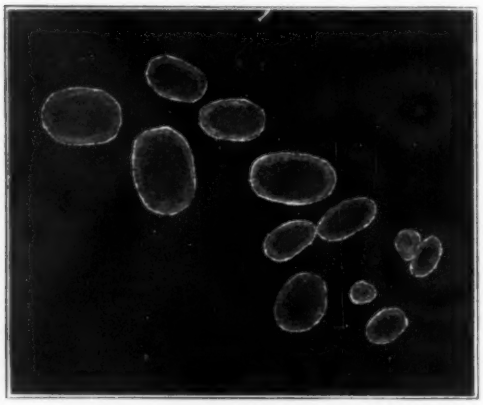

Fig. 10.—A group of the globules seen on the iris of the right eye of IV 47 (R. C., aged 12), three years after discission of the cataract.

IV 48, aged 12, and IV 49, aged 4, were examined ophthalmoscopically, and the media were found to be clear.

IV 50, aged 22; IV 51, aged 21; IV 52, aged 18; IV 53, aged 16; IV 54, aged 12; IV 55, aged 9; IV 56, aged 5; IV 57, aged 2; and IV 58, a baby, all had good eyesight.

IV 59 (fig. 11): B. D., aged 16, had cataracts. Her vision was gradually becoming slightly worse. Refraction, with paredrine hydrobromide-homatropine hydrobromide used as a cycloplegic, showed vision in the right eye with a + 0.50 D. sph. ⌒ — 2.00 D. cyl., ax. 140 to be 6/7.5-2; in the left eye with + 1.00 D. sph. ⌒ — 2.25 D. cyl., ax. 60, 6/7.5 — 3. On retinoscopic examination no zonular opacities were seen, but along the middle half of the lens axis, somewhat radially distributed, were vacuoles and fine granular opacities. Slit lamp examination of the lenses revealed that the individual opacities were smaller than those of her father, III 34, but of the same character. Located in the anterior adult nuclear layer were scattered flakes, the periphery of each iridescent and each center almost clear. Away from the axial region, the opacities were much less dense and appeared as vacuoles. Deeper in this zone the opacities were groups of fine, shiny crystals located at all depths and without zonular distribution. A more dense group of these opacities was located at the position of the posterior Y suture. A few opacities were scattered throughout the center of the lens. Scarcely any opacities were present in the more posterior zones of the lens. The fundi were normal.

IV 60: K. D., aged 8, had refraction performed with atropine used as a cycloplegic. Vision in the right eye with a + 0.25 D. sph. ⌒ + 1.00 D. cyl., ax. 90 was 6/10; in the left eye with a + 0.25 D. sph. ⌒ + 1.00 D. cyl., ax. 75, 6/10. On retinoscopy the outline of the fetal nucleus was visible, but no opacifications were present. By slit lamp, the lenses were clear, except for a few very fine granules in the center of the lens. The fundi were normal.

IV 61: E. B., aged 40, had normal vision.

IV 63: M. B. M., aged 38, had cataracts.

IV 64 was believed to have good vision.

IV 65: R. S., aged 28; IV 66; and IV 67, aged 24, had good eyesight.

IV 68: F. B. M., aged 35, had cataracts. The cataracts had been operated on. She did office work.

IV 70: M. B. T., aged 40, had good vision.

IV 71: W. B., aged 30, did not wear glasses and was believed to have good eyesight.

IV 72: This person, aged 28, had no trouble with the eyes.

IV 73: This person, aged 40, was believed to have good vision.

V 1 (fig. 12): E. H., Jr., aged 12, had cataracts. Vision in the right eye equaled 6/20 + 1; in the left eye, 6/20. The smallest print he could read was Jaeger's test type 6 at 21 cm. with the right eye and Jaeger's test type 5 at 15 cm. with the left eye. The pupils dilated to 7 mm. There was a spherical opacity in the central half of the lens. By slit lamp the fetal nucleus was sharply limited and contained large, semitransparent, circular and oval flakes, each flake centered by a dense white opacity. These were dispersed in greater numbers toward the periphery of the fetal nucleus. In a narrow zone surrounding the nucleus were many minute flakes. Peripheral to this was another zone in which were several groups of posterior opaque fibers. The corresponding anterior fibers were clear. Y sutures were not visible. In the adult nucleus and cortex which ophthalmoscopically appeared clear were visible peripherally very fine, radial, linear opacities, forming riders. The posterior region of the fetal nucleus was readily visible, containing fewer of the large opacities than anteriorly. There was no greater opacity in the center of the fetal nucleus, i. e., the embryonic nucleus.

V 2: L. J., aged 22, in the army; and V 3, D. J., aged 14, both had good eyesight.

V 4: R. F., aged 15, had no visual difficulty.

V 5: R. R., aged 12, had cataracts. Both eyes had been needled before our examination, the right eye unsuccessfully. Evidently the operation on the right eye was followed by uveitis and glaucoma. The left eye had good vision with a cataract lens.

V 6: S. R., aged 11, had refraction performed, with paredrine hydrobromide–homatropine hydrobromide used as a cycloplegic. Vision in the right eye with a + 1.00 D. sph. ⌒ + 1.25 D. cyl., ax. 90 was 6/12; in the left eye with a + 1.50 D. sph. ⌒ + 1.00 D. cyl., ax. 90, 6/7.5 — 2. There was no phoria. The fundi were normal. Slit lamp examination of the lenses revealed them to be of average clearness for her age, except in the upper nasal periphery in the right lens, where there was a small opaque flake limited to a definite zone.

V 7: In M. D., aged 6, and V 8, aged 3, the condition of the eyes was unknown to family.

V 9: In J. D., aged 12, slit lamp examination through dilated pupils showed clear lenses.

V 10: C. D., aged 10, had cataracts. Vision in each eye was 14/200. With pupils dilated to 7 mm. the central half of the lens was a sharply outlined spherical opacity, with a surrounding narrow zone of very fine, powdery spots. Farther out in the lens extending to the periphery were fine riders. In the nuclear opacity, pearl-like spheroids were scattered irregularly in almost the entire outer third. Only occasional spheroids were seen in the deeper zones.

V 11: J. D., aged 5, and V 12, W. D., aged 3, had clear lenses when examined through dilated pupils with slit lamp.

V 13 (fig. 13): B. H., aged 7, had cataracts. She had had difficulty seeing her school work. Vision in the right eye was 18/200, in the left eye 18/200. Retinoscopy with atropine as a cycloplegic was difficult because of the opacity of the lenses, and glasses did not appreciably improve vision. Vision in the right eye

with a —1.00 D. sph. ⌒ —1.50 D. cyl., ax. 180 was 18/200; in the left eye with a —0.25 D. sph. ⌒ and a —0.50 D. cyl., ax. 180, 18/200. The central halves of the lenses were partially opaque spheres surrounded by thin zones of fine punctate opacities. In the center of the lens was a large dense granular opacity surrounded by a zone of fine granules and farther out by a dull milky opaque zone. In the anterior layers of the fetal nucleus were large grayish opaque flakes, each with a white center.

V 14: D. H., aged 6, had cataracts. Retinoscopy through dilated pupils was not difficult. The central, spherical, fetal nucleus had evenly scattered white opacities with a less densely opaque oval halo around each (same as fig. 13 *A*, of V 13). These were evenly dispersed through the anterior part of the fetal nucleus. The embryonic nucleus was no more opaque than the fetal region. The fetal nucleus was surrounded by a single zone of fine, faint opacities. Vision in the right eye with a —0.50 D. sph. ⌒ +3.75 D. cyl., ax. 90

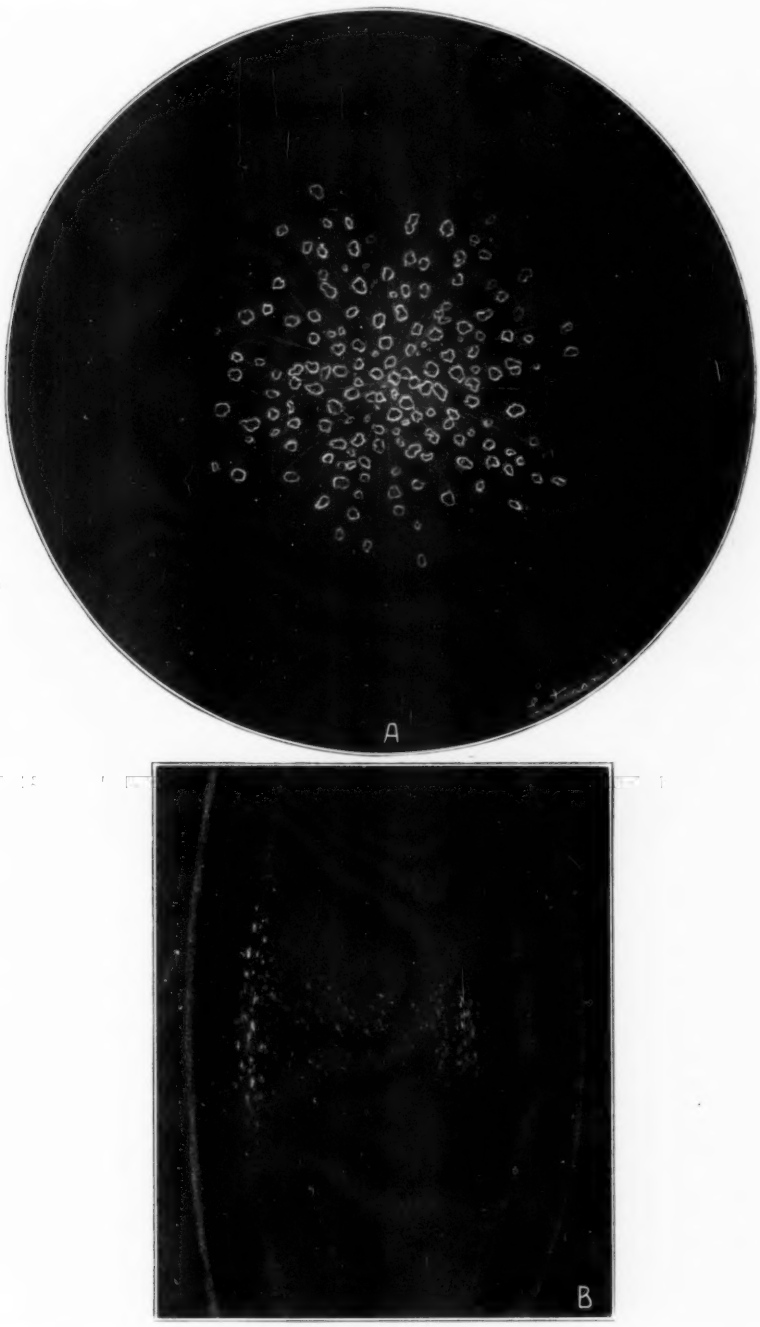

Fig. 11.—*A*, slit lamp view of the cataract in IV 59 (B. D., aged 10). *B*, schematic cross section of the lens in the same person.

was 6/20 — 1: in the left eye with a + 2.50 D. cyl., ax. 90, 6/15 — 1. The fundi were normal.

V 15: In G. H., aged 2, slit lamp examination through dilated pupils showed clear lenses. The fundi were normal. Retinoscopic examination gave the following results: right eye, + 1.50 D. sph. ⌒ + 2.50 D. cyl., ax. 90; left eye, + 1.50 D. sph. ⌒ + 1.25 D. cyl., ax. 90.

V 16: W. C., aged 7, had been examined by an ophthalmologist who did not find cataracts or need of glasses.

V 17: In N. C., aged 5, the lenses examined ophthalmoscopically through dilated pupils were found clear. Slit lamp examination showed three minute white flakes in the right eye just in front of the anterior Y suture, and in the left eye two similar flakes.

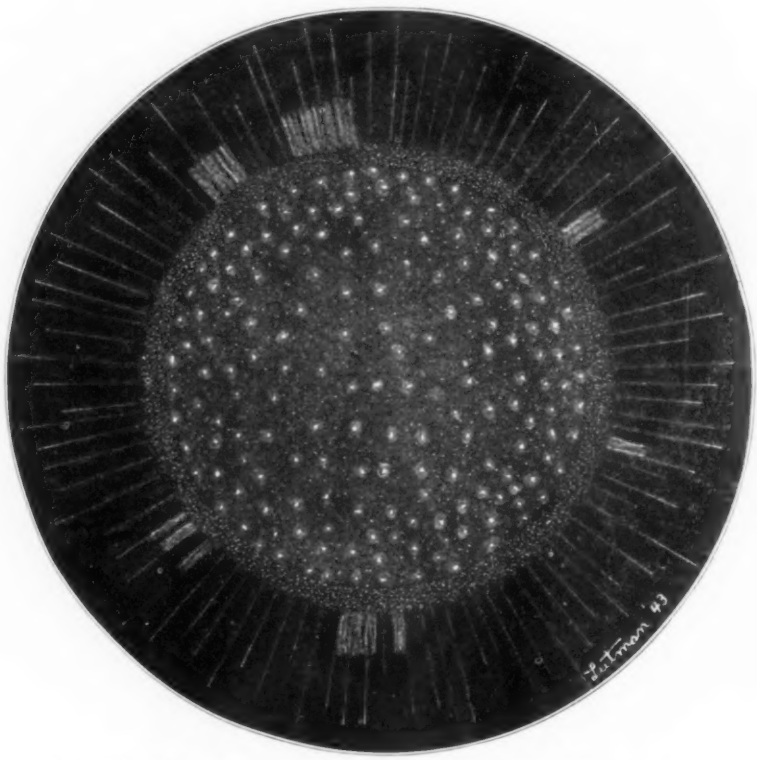

Fig. 12.—Slit lamp view of the cataract in V 1 (E. H., aged 12).

V 18: In T. C., aged 3, the lenses were clear when ophthalmoscopically examined. Slit lamp examination through dilated pupils showed two punctate flakes visible in each eye just before the anterior Y suture.

V 19: M. B., aged 8, had good vision.

V 20: In A. deB., aged 7, the lenses, examined with slit lamp through dilated pupils, showed an occasional minute opacity, but no more than average.

V 21: E. M., aged 13, had good vision.

V 22 and V 23 had no trouble with their eyes.

V 24 and V 25 had no trouble with their eyes.

V 26: B. U., aged 18, had cataracts. Both lenses had been needled.

V 27: G. M., aged 19, had no visual trouble.

V 28: J. M., aged 11, had cataracts. These are believed to have been needled.

V 29: This child, aged 3, is believed to have good eyes.

THE CATARACT

In this genealogy the cataracts fall into three general categories, which are subject to variation and change with age. These three types of cataract are:

(1) Fenestrated, flakelike, iridescent opacities and vacuoles located in the axial portion of the anterior adult nuclear zone, with small shining crystals in the deeper zones. With age, vacuoles and clefts developed in the peripheral cortex and adult nucleus. We saw only 4 members of the pedigree who were afflicted with this type of cataract, II 10 (fig. 3), in whom the fetal nucleus was outlined, and 3 of her descendants—III 30 (fig. 5), III 34 (fig. 6), and IV 59 (fig. 11). In this line of the family no other type of cataract was present.

(2) An opacity of the fetal nucleus. Such cataract occurred in III 18 (fig. 4), with wide variations present in her descendants. In some instances, semiopaque flakes were found in the fetal nucleus, each centered by a dense, white spot, as in V 1 (fig. 12) and V 13 (fig. 13). In other instances (IV 10 and IV 24 [fig. 8, *B*]), distinctly different semitranslucent ovoid opacities were found instead of the flakes. In IV 47 (fig. 10), the spheroid opacities of this last type remained unabsorbed on the iris long after the cataract had been needled. The larger flakes and spheres of the fetal nucleus were more numerous in the anterior and peripheral portions. The

flake opacities became more dense with age. Neither type changed into the other with age, and both were found in young persons. The embryonic nucleus contained more dense opacities in some instances (V 13 [fig. 13]).

Only once, in IV 24 (fig. 8*B*), was the anterior Y suture of the fetal nucleus visible.[12] Here it was accentuated as an opaque Y. The sutures of the embryonic nucleus were not visible in any of the cataracts.

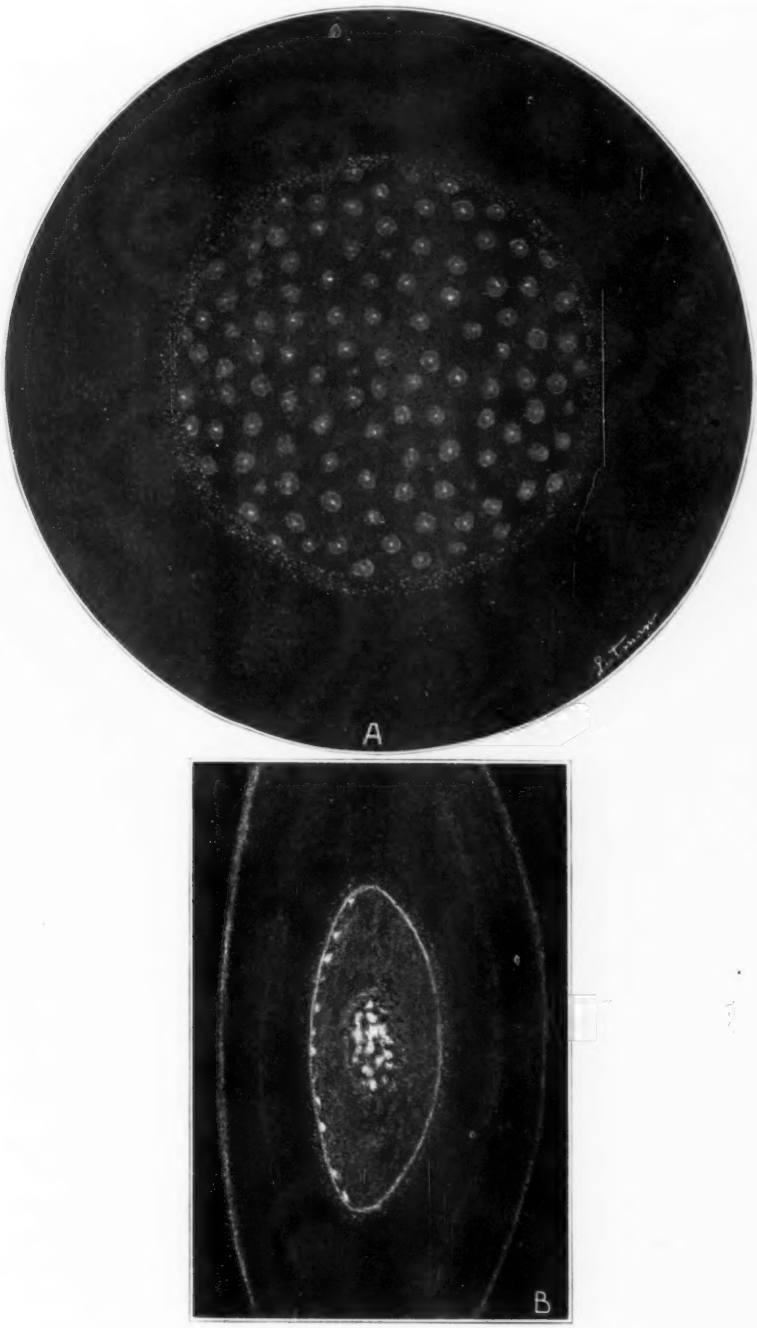

Fig. 13.—*A*, slit lamp view of the cataract in V 13 (B. H., aged 7); *B*, schematic cross section of the lens in the same person.

12. The Y sutures are located on the central embryonic nuclear surface which is surrounded by the peripheral embryonic nucleus (in this paper referred to as the fetal nucleus). The latter nucleus, corresponding to the time just before birth and at birth, also shows a simple suture surface (Vogt, A.: Atlas of the Slit-Lamp Microscopy of the Living Eye, translated by R. von der Heydt, Berlin, Julius Springer, 1921, p. 58).

In several of the adults (IV 18 [fig. 7] and IV 23 [fig. 8*A*]), an anterior polar opacity was present, and continued backward as a fusiform axial opacity to the fetal nucleus. The anterior capsules were normal in all of the cataracts.

Additional zonular opacities occurred in IV 23 (fig. 8*A*) and V 1 (fig. 12), and narrow riders were found in IV 18 (fig. 7), IV 24 (fig. 8*B*), V 1 (fig. 12) and others.

(3) A feather-like cataract formed by opacification of the lens fibers along the anterior and posterior sutures of the adult nucleus. IV 26 (fig. 9) presented such a picture. Except for granular opacities in the embryonic nucleus, no flake opacities were seen in this cataract, which was composed entirely of opaque lens fibers. The children of IV 26 reverted to the fetal nuclear type of cataract (V 13 [fig. 13]).

The fetal nuclear opacity (type 2) caused more visual interference, and more rapid opacification took place, than occurred with the fenestrated flake opacity of the adult nucleus (type 1). Thus II 10 retained useful vision longer than the other members of her generation; her son, III 34, at the age of 36 had better vision than most of the children with a nuclear cataract.

The cataracts in all cases examined which had not been subjected to operative treatment were the same in the two eyes. Anatomically, every zone and surface of the lens is affected by some form of opacification in one or more persons. The most dense opacities tended to form along the lens axis. In 1 instance (II 6) the lens was found to be a complete opacification.

It is significant that these three types of cataract do not appear to be distributed at random throughout the pedigree. For example, type 1 is present in II 10 and all her cataractous descendants whom we examined; III 18 and most of her cataractous descendants, on the other hand, had a type 2 cataract. However, the only person with a type 3 cataract was also a descendant of III 18.

MODE OF INHERITANCE OF THE CATARACT

In any consideration of the inheritance of cataract in this pedigree, the following hypotheses at once arise: 1. All the cataractous persons in this pedigree may have inherited a certain factor which results in the appearance of a developmental cataract, but one or more genetic and possibly environmental modifiers determine to a large extent exactly what type of cataract forms. According to this hypothesis, II 6 and her descendants have inherited, for the most part, a different set of modifiers than, e. g., II 10 and her children, although the "main" factor for cataract is the same in the 2 persons. In other words, the details of the cataract are to a large extent determined by the genetic milieu in which the factor postulated to be responsible for the appearance of a cataract is deposited. 2. A second possibility is that I 1 had two different genetic factors making for cataract, and that some of his children inherited one factor and other children the second factor. 3. Finally, there is the possibility that the gene for cataract present in I 1 mutated to a new allele, and that some affected children received the new allele, the others, the original factor.

A final decision between these possibilities is not feasible at present. However, in view of the rarity of inherited, developmental cataract, it is improbable that I 1 carried two different genes making for this condition. Moreover, mutation to a new allele, although possible, is likewise rare. The first hypothesis therefore emerges as the most probable—if one grants the existence of genetic and possibly unlocalized environmental factors with effects of the magnitudes observed.

In this connection it is of interest to review briefly some of the other pedigrees in which significant variability in the type of cataract has been observed. In the following review we shall quote the authors concerned wherever possible, since, as will be evident, it is not always clear from the terminology exactly what type of cataract is involved.

Knies [6] described a pedigree in which the maternal grandfather, mother and 4 of 6 children had cataract. The cataract of the grandfather is described only as cataracta tremulans and accreta. Concerning the mother he writes that ". . . besonders in der Zone zwischen Kern und hinterer Corticalis, aber auch weniger zahlreich zwischen Kern und vorderer Corticalis eine Masse lichtgrauer Punkte vorhanden waren" (page 216) (". . . especially in the zone between the nucleus and the posterior cortex was a mass of light gray specks, but they occurred also although less numerous between the nucleus and the anterior cortex). The cataracts of the 4 children do not fit into any simple classification. Those of the first child were mainly a combination of nuclear and anterior and posterior polar cataracts (axial); those of the second, nuclear; and those of the third, a combination of nuclear and posterior discoidal. In the fourth child there were significant differences between the two eyes, the right showing a lamellar type and the left an axial cataract.

Gjersing (1878, quoted from Harman [2]) studied a cataractous family in which both nuclear and lamellar cataracts were present, with considerable variation in the exact type of each.

One 4 year old child is reported to have had a "central lenticular cataract in one eye, and diffuse capsular cataract in the other eye" (Harman,[2] p. 164).

Nettleship,[1] in 1906, reviewed the literature on inherited cataract and presented many new pedigrees. The most interesting case in relation to the present considerations is the Betts genealogy (case 58), involving more than 90 persons, 30 of whom were known to be cataractous. This is predominantly a pedigree of coralliform cataract, but in two different instances a child of a father with coralliform cataract had lamellar cataract. In 1 person, son of a cataractous father whose type is not described, the lens showed "opaque pearl-like bodies" toward the center; the periphery was clear. Concerning the possible relationship between coralliform and lamellar cataract, Nettleship writes: "Although . . . it is necessary for descriptive purposes to keep the axial or coralliform apart from the lamellar cataract, it may, perhaps, be found eventually that both arise from the same, or similar, causes operating at different stages, or over longer and shorter periods of foetal life" (p. 217). In addition to this one extensive pedigree, he describes the following shorter family histories, all but one previously unrecorded, in which significant differences were noted in the type of cataract present in parent and in child: (1) Hirschberg's case (1876) of a mother with "complete uncomplicated cataract in one eye, and congenital striopunctate cataract in the other" and her daughter with a double-layered lamellar cataract of both eyes (case 63, page 227); (2) a mother with "small stationary lamellar cataracts" and her son with an "irregular axial opacity from center of lens to posterior pole" (case 74, page 231); (3) a mother with "sharply defined anterior and posterior cortical spokes" and her daughter with "small lamellar cataracts just filling moderately small pupils" (case 76, page 232); and (4) a mother with "cortical cataracts, close beneath anterior capsule, in form of fine dots, and large and small spokes; all parts of surface of cortex affected, except anterior pole, which is quite free" and her son "under care for lamellar cataracts" (case 79, p. 233).

Two years later, Nettleship[7b] described the extensive Coppock pedigree. Of 32 cases of developmental cataract in this group, 24 were of the lamellar type and 8 of the discoid variety, the latter characteristically consisting of a small postnuclear opacity of finely granular appearance. In 2 instances 1 member of a pair of siblings had a lamellar and the other a discoid cataract. Nettleship wrote: "The conclusion seems to be that we have more to learn about the relationship between these two forms of cataract, and that a graduated series will probably be found passing from the smallest and faintest disc of intransparency between anterior and posterior pole, to the common lamellar cataract with its well developed anterior and posterior layers and riders and having a diameter considerably larger than the natural pupil" (p. 231).

Harman[13] has described a family in which "the opacities observed in the lenses . . . were of small size, and vary from complete spherical lamellar opacities of sharply defined contour, to small dense white opacities situated immediately anterior to the lens nucleus, and taking the shape of Y, X or *. The position and general contour of the latter markings suggest the possibility of their being allied to the small forms of the coralliform cataract."[14]

Groenouw,[3] in his treatise on disease of the eye as related to disease elsewhere in the body, was apparently the first to suggest clearly that usually one inherits a tendency to cataract rather than a particular type of opacity: "In der Regel vererbt sich nur die Linsentrübung im allgemeinen, nicht eine besondere Form der Katarakt" (p. 701). (As a rule only the opacity of the lens in general is inherited, not any special type of cataract.) In support of this hypothesis he quoted Hirschberg (1876), Knies (1877), Becker (1883), Kunn (1889), Nolte (1896) and Hosch (1897), but gave no particulars concerning the work of these men. The papers by Hirschberg (1876, quoted from Nettleship[1]) and Knies[6] have already been described. That by Kunn[15] does not contain sufficient data to offer significant support for the hypothesis. We have been unable to verify the other references. Groenouw himself found among the seven children of a father operated on for lamellar cataract two "welche angeborne Trübungen in den vorderen und hinteren Rindschichten der Linse, allerdings nicht von der Form eines typischen Schichtstaares zeitgen" (p. 702). (. . . who showed congenital opacities in the anterior and posterior layers of the lens, but they were not in form of typical lamellar cataract.)

Halbertsma[9] briefly described a pedigree of 95 persons, 92 of whom he examined and 49 of whom had cataract. The defect occurred in two principal forms: "(*a*) Cataracta coerulea punctata ant. et post.; (*b*) perinukleäre Cataracta

13. Harman (footnotes 2 and 8, fig. 307).

14. Harman,[2] p. 142.

15. Kunn, C. G.: Vererbung des Schichtstaares in einer Familie, Wien. klin. Wchnschr. **2**:49, 1889.

zonularis" (p. 108). No further descriptions of the cataracts are given, nor is a detailed pedigree presented.

The pedigree of Welsh and Wegman [10] is comparable to that of Knies [6] in the variety of forms which the cataract may assume. The pertinent observations cover four generations and include 20 persons. The 6 affected males showed an irregular, lamellar type of cataract, associated with tiny white dots in the cortex. In the 6 affected females opacities tended to develop along the posterior embryonic suture and in the posterior Y suture. In some females other defects of the lens coexisted with these opacities. Thus, in 2 persons there were faint central opacities in one lens, while in a third many punctate opacities were scattered throughout the central portion of the lens. The family represented an intermixture of white persons, Negroes and Indians.

In none of these studies can we be certain as to which of the three hypotheses outlined in connection with the present pedigree is correct. However, in view of the rarity of inherited cataract and the infrequency of mutation, it is improbable that any large proportion of these pedigrees represent either mutation or the chance association of two different factors each resulting in cataract. We are therefore led to seek the explanation in the occurrence of genetic or environmental modifiers.

The finding of significantly different types of cataract in the two eyes of one person (Knies,[6] Gjersing, 1878, quoted from Harman [2]) suggests that chance or asymmetrically distributed developmental factors may be important, barring, of course, somatic mutation, which is rare. That genetic modifiers can also create differences of the magnitude observed here is well known from experiments with laboratory animals such as Drosophila, the mouse and the guinea pig. The actual demonstration of the action of genetic modifiers in the human species is difficult because of the impossibility of making the requisite crosses. However, a case in point has been described by Mohr and Wriedt.[16] They studied the inheritance of a type of brachyphalangy in a large family group, finding that the characteristic was inherited as if due to a dominant factor. The malformation occurred in two fairly distinct forms, one more extreme than the other, one form prevailing with few exceptions in one line of the family, and the other form prevailing in another line. They suggest that the difference between the two forms may be due to a dominant modifying factor. The similarity of this case to that described here is striking. Recently Stiles and Pickard [17] briefly described a pedigree of variable malformations of the hands and feet which is perhaps comparable to the one presented here. In none of these examples can we adequately separate the role of genetic and environmental modifiers, but if the latter play an important part they probably exert most of this effect early in the development, i. e., prenatally. As yet little is known concerning the importance to morphogenesis of variations in the intrauterine environment. In the formation of developmental cataract, one must assume that the genetic and environmental modifiers exert their influence to a variable degree throughout the life of the individual, on the developing as well as on the formed portion of the lens.

Assuming, then, that in the present pedigree the occurrence of cataract has the same genetic basis in all affected persons, the question arises as to the type of inheritance involved. It is obvious that here, as in most pedigrees of inherited cataract, a dominant factor is at work. Transmission is direct, from affected to affected. In the entire pedigree there are twenty-four marriages between a normal and a cataractous person, resulting in 79 children: 34 normal, 43 cataractous and 2 of unknown type. This is in agreement with the 1:1 ratio expected from a dominant factor. There are 23 affected males and 20 affected females. Little can be said at present about the nature of the modifying factors which are responsible for the differences between the various types. It is worth pointing out, however, that if the differences between the cataracts of II 6 and II 10 were due to the action of a single, nonlinked autosomal dominant modifier present in II 10 (the simplest genetic hypothesis possible), the chance that her 3 cataractous offspring would inherit this modifier is 1 in 8—a figure well within the bounds of probability. If the differences were due to a sex-linked dominant modifier, then the chance that both the sons of II 10 would receive this factor is 1 in 4, and since any male with such a factor automatically transmits it to his female offspring, IV 59 would of necessity receive the modifier from her father.

COMMENT

In the past it seems to have been for the most part more or less tacitly assumed that a different genetic factor was responsible for each of the almost multitudinous types of developmental

16. Mohr, O. L., and Wriedt, C.: A New Type of Hereditary Brachyphalangy in Man, Publication 295, Carnegie Institution of Washington, 1919.

17. Stiles, K. A., and Pickard, I. S.: Hereditary Malformations of the Hands and Feet, Rec. Genet. Soc. America **12**:55, 1944.

cataract that have been described. It is probable that many different genes do affect the structure of the lens. However, from the foregoing considerations the strong possibility emerges that in at least some of these various pedigrees the "main" genetic factors involved may be the same, with the differences due to the action of modifying genetic and environmental factors. In other words, we are led to a genetic concept of inherited cataract which postulates the existence of a number of different factors whose exact expression is subject to the action of modifying genes, and possibly environmental determinants, rather than to the concept of numerous specific genes each determining a fixed type of cataract. In ethnologically diverse groups the same factor may be expressed differently. If important modifying factors happen to be segregating within a given family, significantly different types of cataract may occur in the members of this family. If no such segregation is occurring, uniformity of type exists—and this happens to be the situation in the majority of pedigrees. In a sense this viewpoint may be regarded as an amplification of the hypothesis advanced by Groenouw in 1920 and quoted by us. It is possible that Duke-Elder [18] had essentially the same thing in mind when he wrote:

> When the intricacy of the architecture of the lens is remembered with its complex lamellar and sutural arrangements, it will be realized that a whole host of morphological types of developmental cataracts with little fundamental distinction are potentially possible. Their essential similarity is emphasized by the fact that in their hereditary transmission one type may replace another in the pedigree (p. 1365).

Considerable has been written, especially by the German authors, concerning the eugenic aspects of inherited cataract, and the possible desirability of sterilizing persons so afflicted. The steady improvement in surgical technics for dealing with this condition makes the considerations of twenty to thirty years ago outmoded. After operation these patients can successfully engage in all but the visually most demanding types of work. As was pointed out in the description of the family, the cataractous members comprising the present pedigree have in no sense been a drag on their community. The society which denies such people as these the right to propagate takes advantage of the presence of a readily recognized and simply inherited characteristic, while ignoring the presence of numerous other equally or more undesirable but less obviously inherited traits whose genetic bases have not yet been worked out. However, it is recognized that the cataractous members of this group may not be typical of persons found in the majority of families with developmental cataract.

18. Duke-Elder, W. S.: Text-Book of Ophthalmology, St. Louis, C. V. Mosby Company, 1938, vol. 2.

SUMMARY

In a genealogy extending over 5 generations and comprised of 123 people, 44 had cataracts from childhood onward. Thirty-four of these 123 persons were examined by the authors: 21 of those examined had cataract.

Three distinct types of cataract can be recognized among the affected persons as follows: (*a*) fenestrated opaque flakes predominantly in the axial region of the anterior adult nucleus, (*b*) a fetal nuclear opacity containing either flakes or spherical bodies with numerous variations in the lens opacities accompanying this, and (*c*) opaque lens fibers, most prominent adjacent to the adult lens sutures. The third type was seen in only a single person.

Type (*a*) was found to the exclusion of other types in one line of the family, while type (*b*) greatly predominated in another branch.

Three hypotheses for the occurrence of distinct types of cataract within a single pedigree are advanced, namely: (*a*) the existence of genetic and environmental modifiers which bring about variations in the expression of a "main" factor, (*b*) the simultaneous presence in the founder of the line of two different, independently inherited factors responsible for cataract and (*c*) the occurrence of mutation.

It is concluded that the bulk of the data, both our own and that in the literature, is best explained by the first hypothesis.

Mrs. James Neel drew the pedigree, and Mr. N. C. Jacobs, medical illustrator, and Mr. M. C. Orser, medical photographer, gave valuable advice in helping us to prepare the figures.

SURGICAL MANAGEMENT OF GLAUCOMA IN CORRELATION WITH GONIOSCOPY AND BIOMICROSCOPY

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The correct surgical management of glaucoma is still an unsettled problem. This paper deals with the limited aid which gonioscopy and biomicroscopy can offer the clinical ophthalmologist in a rational approach to the surgical treatment of glaucoma. It is a summary of our experience with patients of the Meyer Service who have been followed in the glaucoma clinic of the Illinois Eye and Ear Infirmary.

SO-CALLED PRIMARY GLAUCOMA

Acute Glaucoma; Chronic Congestive Glaucoma; Narrow Angle Glaucoma.—The onset of acute glaucoma is manifested by severe pain, with pronounced congestion of the eye and occasional chemosis. Occasionally there may be various degrees of congestion in chronic congestive glaucoma.

Acute primary glaucoma presents a grave ophthalmologic emergency, owing to its fulminating character. Its most important characteristic is the presence of a shallow anterior chamber or a narrow angle. If the chamber is normal or deep, the condition is probably not acute primary glaucoma. Hemorrhagic glaucoma and glaucoma rubrum may first become manifest as an acute attack. However, with these types of glaucoma the anterior chamber is usually deep or normal and the iris is congested. There is also commonly a history of previous decrease in vision. Iritis or uveitis may produce an acute attack, but secondary acute glaucoma must be differentiated from primary glaucoma. In this differentiation gonioscopy and biomicroscopy play an important role. If corneal edema is present and examination of the anterior chamber is difficult, the cornea may be cleared sufficiently for observation by instilling 1 drop of ordinary glycerin, as advocated by Cogan.[1]

The duration of the attack, the visual acuity and the immediate response to intensive therapy with miotics will determine whether one should or should not operate. The choice of a proper type of operation to control the tension within normal limits is important. An attempt should always be made to lower the tension with a miotic before any type of operation is tried, as extremely hard eyes do not respond well to surgical intervention. If there is no response to intensive instillation of a miotic in a few hours and if this absence of response is accompanied with a considerable decrease in visual acuity, operation must be performed immediately.

The biomicroscopic findings are as follows: corneal edema (although this may be absent); a shallow anterior chamber; congestion of the iris; liberation of iris pigment, which may be scattered over the surface of the iris and sometimes over the capsule of the lens, and powdering of the corneal endothelium. Ordinarily the acuity of the angle may be determined with the biomicroscope by placing the slit lamp beam perpendicular to the cornea at the site of the angle of the anterior chamber and the corneal microscope at the angle of specular reflection and focusing it first on the posterior surface of the cornea and then on the anterior surface of the iris.[2]

The gonioscopic findings suggest the term narrow angle glaucoma. Sugar[3] observed that the angle is open, although narrow, at the beginning of the first attack but is closed during the attack and that the extent and firmness of the peripheral anterior synechias vary directly with the duration of the attack.

Surgical Management: The essential evidence obtained with the biomicroscope and the gonioscope is that the angle of the anterior chamber is narrow, and the acuity of the angle depends largely on the extent of the peripheral synechias anteriorly. The surgical problem is, therefore, to enter the anterior chamber with minimum danger to the iris and the capsule of the lens and to avoid sudden decompression of an eyeball with a high intraocular pressure.

This can be accomplished by making a scratch incision or an incision from the outside after a conjunctival flap has been dissected to the limbus.

From the Illinois Eye and Ear Infirmary.

Read at the Forty-Ninth Annual Meeting of the American Academy of Ophthalmology and Oto-Laryngology, Chicago, Oct. 11, 1944.

1. Cogan, D. C.: Clearing of Edematous Corneas by Glycerin, Am. J. Ophth. **26**:551, 1943.

2. Sternberg, P.: To be published.

3. Sugar, H. S.: Concerning the Chamber Angle: I. Gonioscopy, Am. J. Ophth. **23**:853, 1940.

The scratch incision, made with either a Lundsgaard knife or a keratome, can be beveled to any degree in order to enter an anterior chamber with an extremely narrow angle. The incision may be enlarged with corneal scissors. This type of incision provides for slow decompression, and ordinarily prolapse of iris tissue occurs; also, with this type of incision one avoids entering the chamber with sharp instruments. An incision may be made with a small keratome or a Graefe knife if preferred, provided the anterior chamber is not extremely shallow. Retrobulbar injection of 1 cc. of 2 per cent solution of procaine hydrochloride with a few minims of a solution of epinephrine hydrochloride (1:1,000) is always given just prior to all surgical operations for glaucoma, but this dose is not always adequate for sufficient lowering of the tension. One should always wait five to ten minutes for adequate anesthesia and a possible decrease in tension.

Iridencleisis is to be preferred to the classic basal iridectomy of von Graefe [4] as the treatment for narrow angle glaucoma. The rationale of iridectomy is still a matter of much controversy. It is generally held, as originally suggested by Weber,[5] that the main effect is the mechanical opening of a closed angle and that the operation is successful only in so far as it accomplishes this and if the iris is removed so completely that blocking of the angle cannot recur. However, the inflammatory reaction, together with contact of the iris tissue with the walls of the angle during the immediate postoperative period before the shallow anterior chamber has reformed, may contribute to additional peripheral anterior synechias. Kronfeld [6] stated that the smallest sector of open angle compatible with normal intraocular pressure is 70 to 90 degrees of arc.

The mechanical concept does not furnish the complete explanation, for gonioscopic examination has shown that the results are successful in many cases if the base of the iris is left and is poor if the base is totally removed (Uribe Troncoso [7]; Werner [8]). Among many other explanations, these suggestions have been made: The vasomotor reflexes are abolished (Abadie [9]); a filtering scar is formed accidentally, with or without the incorporation of uveal tissue (de Wecker [10]); since a wound in the iris does not cicatrize (Fuchs [11]), a permanent fresh absorbing surface is exposed to the aqueous.

Since the rationale of iridectomy is controversial and since it has been shown by Uribe Troncoso,[7] Thorburn,[12] Werner,[13] Barkan [14] and McLean [15] that the peripheral anterior synechias are a result, and not the cause, of glaucoma and that, therefore, the cause of narrow angle, or acute congestive, glaucoma is not entirely "mechanical," the factors involved in the other types of primary glaucoma may be prevalent here as well. It seems ill advised, therefore, to depend on the mechanical concept of making the normal drainage channels available through iridectomy. True, iridectomy is frequently successful, but we believe iridencleisis is the more logical procedure. Basal iridectomy, if preferred, should be done during the first attack of acute glaucoma and if the attack is of less than twenty-four hours' duration.

The narrow angle and the effects of the narrow angle offer a sufficient mechanical hazard during operation and afterward to make trephination a poor choice. It is necessary that the trephine opening be placed sufficiently anteriorly in the cornea to avoid the formed peripheral anterior synechias and ciliary incarceration, and this in itself is difficult. In attempts to do this, a flap consisting not only of the corneal epithelium but of the epithelium and the corneal stroma is dissected; when the flap is replaced after the trephination, corneal stroma becomes firmly anchored to corneal stroma, with prompt closure of the trephine opening. The technical difficulties and the accidental injuries which can occur with the trephination need no mention.

Chronic Simple Glaucoma; Glaucoma Simplex; Wide Angle Glaucoma.—Glaucoma simplex is characterized by certain changes in the visual fields, increased intraocular tension, especially

4. von Graefe, A.: Ueber die Iridectomie bei Glaucom und über den glaucomatösen Process, Arch. f. Ophth. **3**(pt. 2):456, 1857.

5. Weber, A.: Die Ursache des Glaucoms, Arch. f. Ophth. **23**(pt. 1):1, 1877.

6. Kronfeld, P. C.: In Search of Gonioscopic Correlates of Responsiveness to Miotics, to be published.

7. Uribe Troncoso, M.: Gonioscopy and Its Clinical Applications, Am. J. Ophth. **8**:433, 1925.

8. Werner, S.: Gonioscopic Studies on Glaucomatous Eyes Following Operation, Finska läk-sällsk. handl. **73**:981, 1931.

9. Abadie, C. H.: Section de la zone ciliaire ou ciliarotomie, Arch. d'opht. **30**:262, 1910.

10. de Wecker, L.: Traité théorique et pratique des maladies des yeux, Paris, A. Delahaye, 1867, vol. 2, p. 571.

11. Fuchs, E.: Ber. ü. d. Versamml. d. ophth. Gesellsch. **25**:179, 1896; cited by Duke-Elder, W. S.: Text-Book of Ophthalmology, St. Louis, C. V. Mosby Company, 1941, vol. 3, p. 3401.

12. Thorburn, A.: A Gonioscopic Study of Anterior Peripheral Synechiae in Primary Glaucoma, Thesis, Stockholm, 1927.

13. Werner, S.: Gonioscopical Studies of Operated Cases of Glaucoma, Acta ophth. **10**:112, 1932.

14. Barkan, O.: New Operation for Chronic Glaucoma: Restoration of Physiological Function by Opening Schlemm's Canal Under Direct Magnified Vision, Am. J. Ophth. **19**:951, 1936.

15. McLean, J.: Gonioscopy in Glaucoma Operations, Tr. Am. Acad. Ophth. **45**:176, 1941.

during the early morning hours, and various degrees of cupping of the optic disk. If these changes cannot be held in check with the use of mild miotics, surgical treatment is indicated.

The biomicroscopic findings ordinarily do not differ from the normal unless vascular decompensation occurs. Varying degrees of atrophy (of the iris), however, may be present.

The gonioscope reveals exclusion of the narrow angle mechanism; hence the term wide angle glaucoma. Other gonioscopic findings are deposits, of varying amounts of pigment, within the corneoscleral trabecula; decreased translucency of the trabecula, or trabecular sclerosis, and peripheral anterior synechias in late stages. Kronfeld, McGarry and Smith [16] observed an abnormal filling phenomenon of Schlemm's canal after paracentesis of the anterior chamber or massage of the eyeball.

Surgical Management: The successful surgical management of wide angle glaucoma depends on the functional efficacy of new outflow channels. The presence of peripheral anterior synechias before or after operation does apparently alter the efficacy of these new outflow channels to some extent.[15]

Iridencleisis is preferred except when atrophy of the iris is present. This is a much less difficult procedure technically and can be done with less chance of accidental damage to important structures. It is, therefore, more applicable to use by the average as well as by the expert operator.

However, trephination may be done if it is preferred, since with wide angle glaucoma there is "sufficient room" in the angle for the procedure. It is definitely the operation of choice when atrophy of the iris is noted.

Cyclodialysis will be discussed subsequently.

GLAUCOMA FOLLOWING REMOVAL OF CATARACT

This form of glaucoma was shown by Czermak [17] to result from peripheral anterior synechias due to delayed restoration of the anterior chamber after cataract extraction. This cause was confirmed gonioscopically by Sugar [3] and by Kronfeld and Grossman.[18] The extent and the firmness of the peripheral anterior synechias vary directly with the duration of the contact of the iris root with the wall of the angle.

The gonioscope reveals closure of the angle, with peripheral anterior synechias of varying extent. Kronfeld and Grossman [18] stated the belief that the blue or gray iris forms synechias characterized by fuzzy edges, while the brown iris with the dense anterior border forms synechias with solid line edges, as viewed with the gonioscope.

Surgical Management: Although in some cases this form of glaucoma may respond well to a miotic, tolerance to the drug seems to occur within a relatively short time, and surgical treatment will finally be required in many cases.

With regard to cyclodialysis, Barkan [19] and Sugar [3] noted an "all or none" relation between the success of the operation and the formation of a communication between the anterior chamber and the supraciliary space, which can be seen as a definite cleft with the gonioscope. McLean [15] showed that the success of the cyclodialysis varies directly with the acuity of the angle and the depth of the anterior chamber. Therefore, cyclodialysis is usually not advocated for narrow angle glaucoma and is advised only on occasion for wide angle glaucoma, particularly when associated with low elevated tension, or as a secondary operative procedure for wide angle glaucoma with slightly elevated tension following a previous partially successful filtering operation. Its greatest efficacy for glaucoma is after removal of a cataract. Many well performed cyclodialyses fail because of the organization of a blood clot in the cleft. The avoidance of the horizontal meridian, where the posterior ciliary vessels and nerves are located, and the use of physostigmine after operation will minimize this complication. Observance of Gradle's [20] suggestion that the operation be done in one of the upper quadrants and that the patient sit up in bed after operation will tend to keep blood out of the cleft if hemorrhage occurs. The limbs of the coloboma should be avoided to prevent unnecessary trauma and hemorrhage. If cyclodialysis fails, it may be repeated. If it fails again, trephination may be tried.

A means of preventing formation of peripheral anterior synechias, with delayed restoration of the anterior chamber after cataract extraction, is the use of an adequate corneoscleral suture at the time of the cataract extraction.[15] Another means is postoperative injection of air into an unrestored chamber, as advocated by McLean,[15] MacMillan [21] and others.

16. Kronfeld, P. C.; McGarry, H. L., and Smith, H. E.: Gonioscopic Studies on the Canal of Schlemm, Am. J. Ophth. **25**:1163, 1942.

17. Czermak, W.: Die augenärzliche Operationen, Vienna, C. Gerold's Sohn, 1898.

18. Kronfeld, P. C., and Grossman, E. E.: The Relation of the Gonioscopic Findings to the Incidence of Secondary Glaucoma in Operative Aphakia, Tr. Am. Acad. Ophth. (1940) **45**:184, 1941.

19. Barkan, O.: Mode of Action of Cyclodialysis, Am. J. Ophth. **19**:21, 1936.

20. Gradle, H. S.: Personal communication to the author; cited by Sugar.[3]

GLAUCOMA ASSOCIATED WITH CAPSULAR EXFOLIATION

Glaucoma is not present in all eyes with senile capsular exfoliation; hence, the term glaucoma associated with capsular exfoliation is preferred, rather than glaucoma capsulare.

The biomicroscope reveals white, crustlike flakes as a central disk on the anterior surface of the lens, corresponding in extent to a minimum dilatation of the pupil, and as a granular girdle about the periphery, with a clear band between the two areas and deposition of these flakes on the iris and cornea.

The gonioscope shows depositions of these fine flakes throughout the structures of the angle, usually accompanied with heavy pigmentation of the trabecula. The angle is of the wide type, with relative absence of peripheral anterior synechias.

Surgical Management.—Successful surgical management depends on producing new channels of outflow. Iridencleisis or trephination may be done, since the angle is usually wide. Iridencleisis is preferred for the same reasons as those obtaining in treatment of wide angle glaucoma.

When the lens is sufficiently opaque to warrant cataract extraction and the glaucoma is not controlled with use of miotics, it is advisable to do the operation for glaucoma first. After the eye has completely recovered, the operation for cataract may be performed, with extraction of the lens through an incision placed below, temporally or anteriorly in the cornea, provided the antiglaucoma bleb does not overhang too far anteriorly on the cornea.

HEMORRHAGIC GLAUCOMA

Hemorrhagic Glaucoma Following Occlusion of the Central Retinal Vein.—The biomicroscope reveals neovascularization of the iris, in addition to many or all of the features of acute congestive glaucoma.

The gonioscope reveals an open angle at first, with considerable congestion and neovascularization of the root of the iris. Somewhat later the angle becomes completely closed.

The surgical management will be considered along with that of diabetic rubeosis iridis.

Diabetic Rubeosis Iridis.—Biomicroscopically and gonioscopically the appearance of rubeosis iridis diabetica is similar to that of glaucoma following obstruction of the central retinal vein. The angle is closed and covered with formations of fine new vessels.

Surgical Management.—For this type of glaucoma, we prefer cyclodiathermy puncture of the ciliary body, as described by Vogt.[22] The inferior half of the pars corona of the ciliary body is treated with diathermy. The tension is lowered, although not always to within normal limits, but freedom from pain nearly always occurs within thirty-six hours after the operation. Because of the scleral "shrinkage," with resultant decrease in the total volume capacity of the eyeball, which follows the diathermy, extensive diathermy over the complete circumference of the pars corona may raise the intraocular pressure during the immediate postoperative period above the systolic blood pressure in the central retinal artery, with resulting occlusion of the artery; this occurred in 1 case at the infirmary. No case of sympathetic ophthalmia has occurred as a result of this procedure in well over 100 cases at the infirmary in which cyclodiathermy was performed.

Enucleation is, of course, the alternative surgical procedure.

GLAUCOMA ASSOCIATED WITH LENTICULAR INTUMESCENCE

Swelling of the lens produces narrowing and later actual blockage of the angle by the root of the iris.

The biomicroscopic and gonioscopic pictures are the same as those for acute glaucoma except for the lenticular intumescence.

Surgical Management.—The surgical management resolves itself into removal of the lens. This is accomplished after the chamber is entered by means either of a scratch incision or of an incision made with a small keratome and enlarged with scissors. In this manner one avoids sudden decompression as well as traumatic injury to the iris or lens.

SUMMARY

Gonioscopy and biomicroscopy can be of limited aid in the surgical management of the various types of glaucoma. These clinical instruments present evidence with which the ophthalmologist may better evaluate the type and site of the glaucoma and determine a rational surgical approach to the glaucoma in the individual case.

58 East Washington Street.

21. MacMillan, J. A.: Air Injection as a Factor in Maintaining Filtration Following Corneo-Scleral Trephining in Glaucoma, Tr. Am. Ophth. Soc. **37**:127, 1939.

22. Vogt, A.: Versuche zur intraocularen Druckherabsetzung mittelst Diathermieschädigung des Corpus ciliare (Cyclodiathermiestichelung), Klin. Monatsbl. f. Augenh. **97**:572, 1936.

PATHOLOGIC PHYSIOLOGY OF CONVERGENT STRABISMUS

MOTOR ASPECTS OF THE NONACCOMMODATIONAL TYPE

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I accepted with both reluctance and pleasure the invitation to deliver this the first Gifford Memorial Lecture. You can appreciate how difficult it is to prepare a lecture adequate for this occasion, for Sanford Gifford will always live in our memories through his own contributions to our literature.

He wrote with a profound factual knowledge, with the intuition and acumen of a skilled investigator and with a clinical wisdom which comes only from a rare combination of intelligence and common sense—in short, his writings show all the qualities of genius and will themselves constitute a splendid and an enduring memorial.

None of us, I am sure, could ever feel competent to add luster to his fame by any of our own works, but all of us would be most grateful for an opportunity to add our tribute, however small, to the memory of one for whom we had such great affection and high esteem. It is for this opportunity that I thank you most sincerely.

Convergent strabismus is not a single entity produced by one etiologic factor but is an abnormal position of the visual axes due to a number of different causes. Therefore no single theory of the cause of convergent strabismus satisfactorily explains all of the cases one sees.

Donders' accommodative theory fits the largest number; in some of the cases the squint is undoubtedly of paralytic origin, but many others do not fall into either of these groups. Worth's conception of congenital absence or failure of development of a fusion sense as a fundamental factor in the causation of squint conflicts with the fact, widely recognized today, that many of the patients acquire some degree of fusion, and a few even develop stereopsis, after their eyes have been straightened. In addition to this, his theory still leaves unanswered the important question of what causes the abnormal convergence of the visual axes. It will be remembered that Worth insisted that there were two essential conditions present in every case of comitant convergent squint: (1) "an abnormal convergence of the visual axes; (2) a defect of the fusion faculty."[1]

The stress which Chavasse[2] laid on the development of the normal reflex association of the eyes has focused attention on squint as being a perversion of these reflexes, due to any obstacle which dissociates the eyes; and to this I heartily agree. He enumerated some of the motor and sensory obstacles which might prevent or break up the association of the two eyes in the period in which these reflexes are forming. He stated the belief that in many of these cases the squint is due to paralysis of ocular muscles which have recovered.

TABLE 1.—*Classification of 232 Cases of Convergent Squint According to Cause*

Type of Squint	No. of Cases	Percentage
Accommodative		
A. Completely	62	27
B. Partially	69	29
Paralytic	34	15
Undetermined	67	29
Total	232	100

If one leaves out the cases in which squint is the result of paralysis of ocular muscles, all squints are due fundamentally to an abnormal convergence innervation. The group of accommodative squints is the best example of this. It is not necessary to recite the mechanism by means of which an abnormal amount of hyperopia causes squint during the early formative years, by the excessive convergence impulses.

But in only about a third of the cases one sees is the squint wholly accommodative. In table 1 are classified 232 cases of convergent squint from the records of my private cases according to the causative factor.

In about one third of these cases the squint was of undetermined origin. Cases were put in this group only after it was apparent that they could not fit into one or the other of the groups;

From the Hospital of the University of Pennsylvania.

The first Gifford Memorial Lecture, delivered before the Chicago Ophthalmological Society, Jan. 15, 1945.

1. Worth, C.: Worth's Squint, ed. 5, Philadelphia, P. Blakiston's Son & Co., 1921.

2. Chavasse, in Worth,[1] ed. 7, 1939.

i. e., there were no signs of past or present paralysis of ocular muscles; the angle of squint was the same for distance and for near vision; glasses did not change the angle of squint, and the amount of hyperopia present was normal for the age.

In the majority of these cases there was complete disappearance of the squint when the patient was under anesthesia. The operative notes show that in many instances the eyes were divergent under the anesthesia before the operation was commenced. This is also true of the other types of squint, accommodative and, in some instances, paralytic, but this fact does not alter the conclusion which one must draw; i. e., that these squints, like the accommodative type, are due to an abnormal convergence innervation. The only difference between the two forms is that in one the source of the abnormal convergence innervation is known, i. e., excessive accommodation, whereas in the other form one is as yet totally ignorant of where the abnormal convergence comes from or what it is due to.

If one inquires into the possible sources of abnormal convergence tone, one finds that knowledge of the mechanisms by means of which the ocular muscles are supplied with normal tonic impulses is very sketchy.

It is the primary purpose of this paper to review the normal physiologic mechanism which coordinates the movements of the eyes, in the hope that it will throw some light on the etiologic factors in this large group of cases. For purposes of description, the voluntary movements will be dealt with separately from the involuntary, or reflex, movements, but it should be understood that the two mechanisms operate simultaneously and harmoniously.

VOLUNTARY CONTROL OF OCULAR MOVEMENTS

Voluntary movements of the eyes occur as the result of incoming sensory impressions which give rise to a conscious desire to change the position of the eyes, or as the result of impressions that have been received in the past and stored away in the areas of the cortex having to do with memory. The latter, when they reach the level of consciousness, again call for volitional movement. Willed movements are usually purposeful ones and call into play many mechanisms which must be coordinated. Whatever the incentive may be to execute a coordinated muscular movement, it starts in the motor cortex as an excitation of the large pyramidal cells of the precentral convolution, the so-called Betz cells.

Cortical Centers for Lateral Gaze.—In the case of willed ocular movements the pyramidal cells of the caudal extremity of the second, and possibly the third, frontal convolutions of each hemisphere initiate the action. According to Penfield and Erickson,[3] stimulation of a zone which centers on Vogt's area 8 (alpha, beta, gamma), shown in figures 1 and 2, causes conjugate deviation of the eyes to the opposite side and often upward deviation. These movements result from mild stimulation which does not cause convulsive movements. Movements, and

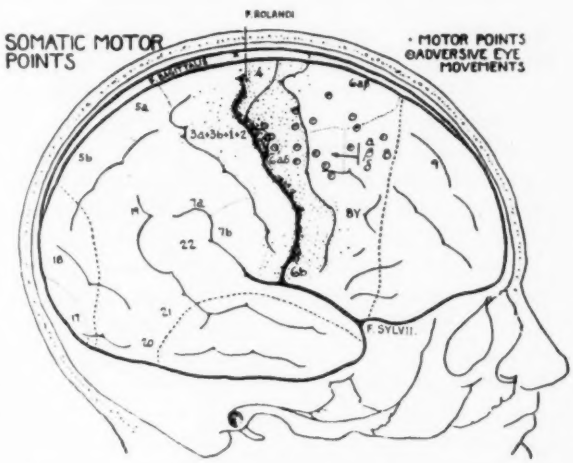

Fig. 1.—Surface view of the right motor cortex, showing the areas where stimulation in the conscious subject produced conjugate lateral deviation to the left. Occasionally upward movements were observed. From Penfield and Erickson.[3]

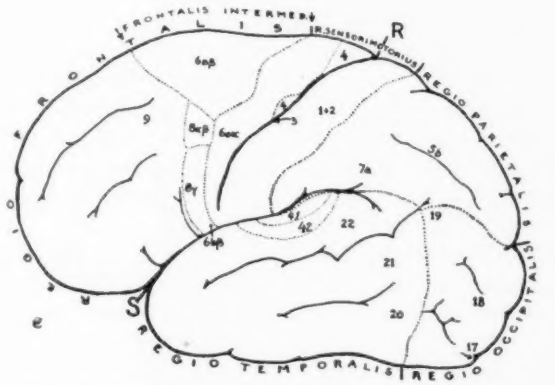

Fig. 2.—Surface view of the left motor cortex, slightly tilted to show Vogt's areas 8 α, β and γ. These are the areas for voluntary ocular movements to the right, and possibly upward. From Penfield and Erickson.[3]

No cortical areas have been discovered where stimulation produces either downward movements or movements of convergence.

not individual ocular muscles, are represented in the cortex. With the exception of the muscles

3. Penfield, W., and Erickson, T. C.: Epilepsy and Cerebral Localization, Springfield, Ill., Charles C Thomas, Publisher, 1941, p. 45.

which innervate the lids, there is no isolated representation of any of the ocular muscles in the cortex.

Stimulation of the second frontal convolution in the higher mammals usually produces conjugate deviation of both eyes to the opposite side, but the early experiments of Russell and Sherrington [4] showed that after section of various groups of the ocular muscles stimulation of the second frontal convolution on one side might lead to conjugate deviation of the eyes in almost any direction, depending on the combination of the muscles which were made ineffective by sectioning; so they concluded that this region was responsible for voluntary ocular movements in all directions, laterally and vertically. They argued that since lateral movements are so much more commonly exercised, these movements overshadow the control of the other conjugated movements. Stimulation of the right frontal convolution usually produces conjugate deviation of the eyes to the left side, and stimulation of the cortex of the left frontal convolution produces deviation of the eyes to the right side. Destruction of either area produces paralysis of conjugate movement of the eyes toward the side of the lesion, without impairing the action of the individual muscles concerned and without producing diplopia, since the limitation of movement affects both eyes equally and simultaneously.

Cortical Centers for Vertical Gaze.—Although all investigators are agreed on the cortical origin of lateral movements, there is no such unanimity on the origin of the movements of vertical gaze. The experiments of Russell and Sherrington [4] and those of Grünbaum and Sherrington [5] indicated that upward deviation may be produced by stimulating either frontal area, while Duke-Elder [6] claimed that stimulation of the upper part of either frontal convolution produces downward movement and stimulation of the lower part produces upward movement of the two eyes.

Since there is often a gradual recovery of function after lesions which completely prevent conjugate deviation of the eyes to one side, it is probable that there is some degree of bilateral innervation for conjugate lateral movements.

Course of Fibers to the Pontile Center.—The fibers which arise from the pyramidal cells of the second frontal convolution pass into the corona radiata of the white matter and enter the knee of the internal capsule as part of the pyramidal tract (fig. 3). They turn into the internal capsule close to the fibers from the cortical representation of the motor innervation of the face and proceed downward into the pons. Separate contingents of this tract are given off at different levels, the chief of which is that which has to do with vertical gaze, i. e., the fibers innervating the muscles of upward and downward gaze, together with the muscles of the upper eyelid and the frontalis muscle. These fibers leave the pyramidal tract at about the level of the superior corpora quadrigemina, where they probably go to the center for vertical gaze (see page 366).

If there are fibers in this system which have to do with convergence, their presence has never been determined, although convergence movements have been produced by stimulation of the center in the frontal lobe (Riley [7]) and can be initiated volitionally.

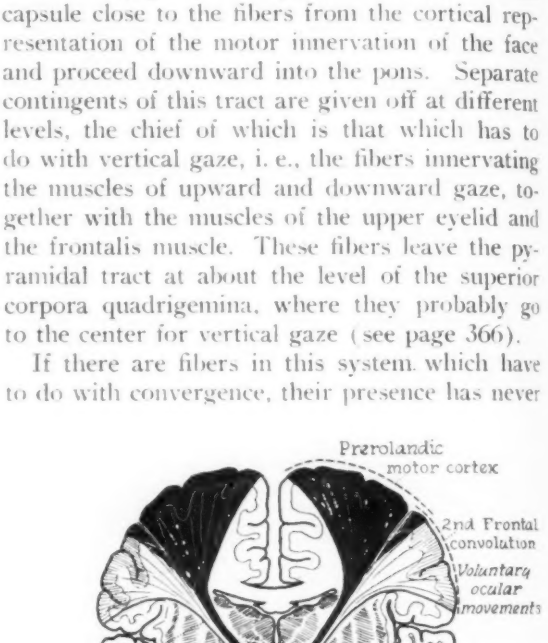

Fig. 3.—Diagram of the course of the voluntary oculogyric pathways from the frontal cortex to the pons. The fibers enter the knee of the internal capsule as part of the pyramidal tract. In their course downward separate contingents are given off at different levels, the chief of which is that which concerns vertical gaze.

According to Spiegel and Sommer,[9] these fibers run into the vestibular nuclei on their way to the hypothetic center for lateral gaze in the pons.

The part of the pyramidal tract which has to do with lateral gaze leaves "the corticonuclear tract as two bundles of fibers, the pes lemniscus superficialis and the pes lemniscus profundus in the midbrain and upper pontile region, respec-

4. Russell and Sherrington, cited by Riley.[7]

5. Grünbaum, A. S. F., and Sherrington, C. S.: Observations on the Physiology of the Cerebral Cortex of Some of the Higher Apes, Proc. Roy. Soc. London **69**:206, 1901-1902.

6. Duke-Elder, W. S.: Text-Book of Ophthalmology, St. Louis, C. V. Mosby Company, 1933, vol. 1, p. 207.

7. Riley, H. A.: The Central Nervous System Control of the Ocular Movements and the Disturbances of This Mechanism, Arch. Ophth. **4**:640 (Nov.); 885 (Dec.) 1930.

tively; descends for a short distance in the mesial fillet and then proceeds dorsally in the tegmentum to the center for lateral movement" (Riley [7]).

Besides conjugate deviation in a horizontal or vertical direction, stimulation of the frontal centers in the lower mammals may produce ocular torsion (Collins and Spiegel [8]). In cats the most frequent result is intorsion of the opposite eye, while the eye on the side stimulated intorts or extorts or does nothing at all. No tonic effect of the cortex on torsion could be found, according to these authors, and elimination of the cortex and subcortical ganglions in front of the midbrain produced only moderate degrees of torsion. Under the influence of anesthetics, however, particularly the so-called brain stem anesthetics, pronounced torsion was observed. The range of torsion was from 35 degrees inward to 30 degrees outward. There was mostly bilateral intorsion, rarely bilateral extorsion or conjugate deviation.

An analysis of this effect of barbiturates showed that it was only partially due to depression of prosencephalic and diencephalic activity and partly to direct action upon the midbrain and upon rhombencephalic supranuclear centers (probably vestibular nuclei).[9]

The Pontile Center.—It is generally accepted that the fibers for voluntary lateral gaze cross over to the opposite side of the brain stem just above the level of the sixth nerve nuclei. From here their course is not so certain. The majority of authors favor the assumption that the fibers end in a pontile center for lateral gaze, although there is no histologic evidence of such a center. Its existence is based on the fact that in certain cases in which the nucleus of the sixth nerve is entirely destroyed on one side, there is loss of movement of the homolateral eye outward but retention of the conjugate movement of the opposite eye toward the side of the lesion. This proves conclusively that the nucleus of the sixth nerve itself cannot be the center for lateral movement but that either there is a separate center outside the nucleus or the fiber splits before reaching the nucleus of the sixth nerve and sends one part to the homolateral nucleus of the sixth nerve and another to the contralateral nucleus of the internal rectus muscle. Most authors have asserted that there is a separate center but that it lies close to the nucleus of the sixth nerve (Holmes,[10] Kidd [10]), probably in front of the nucleus and nearer the midline. This so-called pontile center for lateral gaze receives all of the stimuli which excite horizontal conjugate movements of the eyes, i. e., not only the voluntary impulses previously described but the reflex innervation from the lower visual and auditory systems, proprioceptive impulses from the neck muscles, and impulses from the visuomotor center in the occipital cortex.

The Posterior Longitudinal Bundle.—From this center the fibers run into the posterior longitudinal bundle, the fiber tract which links up the nuclei of the third, fourth and sixth cranial nerves. Spiegel and Sommer [11] stated the belief that the corticofugal impulses enter the posterior longitudinal bundle through the mediation of the vestibular nuclei, for lesions of these nuclei, as well as physiologic changes in the state of their excitability, essentially alter the effect of stimulating the cortical centers for conjugate movements of the eyes (fig. 4). This view, according to these authors, explains how the law of reciprocal innervation (inhibition of antagonists during excitation of a muscle or group of muscles) acts on stimulation of the cortical centers. It was

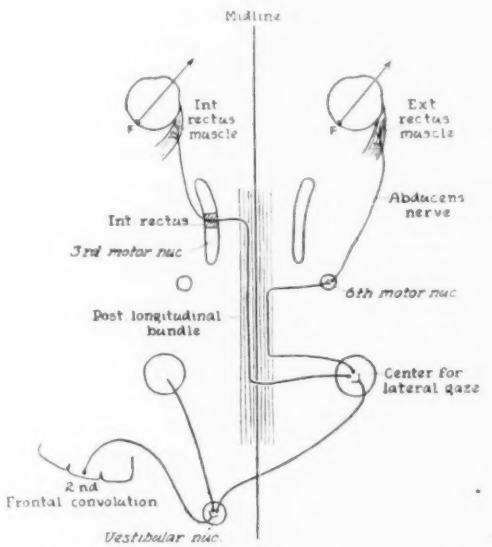

Fig. 4.—Diagram of the pathway for voluntary right lateral gaze. Starting in the second frontal convolution, the fibers run down in the pyramidal tract to the pons, where they enter the vestibular nuclei, and then to both centers for lateral gaze, near the nuclei of the sixth nerve. This pathway links the voluntary impulses for lateral gaze with the involuntary impulses entering through the vestibular nuclei (see fig. 14).

8. Collins, D. A., and Spiegel, E.: Ocular Rotation: Influence of Anesthetics and Operations on Various Parts of Central Nervous System, Proc. Soc. Exper. Biol. & Med. **39**:100 (Oct.) 1938.

9. Spiegel, E. A., and Sommer, I.: Neurology of the Eye, Ear, Nose and Throat, New York, Grune & Stratton, Inc., 1944, p. 390.

10. Holmes, G.: Palsies of Conjugate Ocular Movements, Brit. J. Ophth. **5**:241 (June) 1921. Kidd, L. J.: Fourth Cranial Nerve, ibid. **6**:49 (Feb.) 1922.

11. Spiegel and Sommer,[9] pp. 393-394.

shown by Sherrington in experiments on monkeys that one could stimulate the right second frontal convolution and produce conjugate deviation of the eyes to the left even after the muscles which produce levoversion in the left eye have been severed. Under such circumstances, this movement of the left eye outward could occur only by relaxation of the left internal rectus muscle. Cortical stimulation, therefore, not only produces contraction of the ocular muscles which move the eyes conjugately to the opposite side but simultaneously produces relaxation (inhibition) of their antagonistic muscles. This action has been shown to occur also in man.[12] Spiegel pointed out that this action can occur by reason of the fact that the vestibular nuclei act as a relay station which sends fibers into both ipsilateral and contralateral posterior longitudinal bundles.

Once the impulses for horizontal conjugate movements get into the posterior longitudinal bundle, they innervate in an appropriate fashion the nuclei of the ocular muscles necessary to produce the associated movements of the two eyes. Like all voluntary movements, these coarse stimuli must be refined and integrated by reflex activities from all the sources that control the "associated, synergic and voluntary control over the oculomotor movements" (Riley), and these moderating impulses are converged on the neuromuscular apparatus through the medium of this bundle.

Centers for Vertical Gaze.—Still less is known of the subcortical centers and pathways controlling voluntary vertical movements of the eyes. The question of the localization of these movements in part of the frontal oculogyric center has already been discussed. Movements of upward and downward gaze must be associated with simultaneous stimulation or inhibition of the muscles which raise and lower the lids. Lesions in the neighborhood of the superior colliculus have long been known to be associated with disturbances in vertical gaze and with changes in the movements or position of the lids, such as ptosis and tucked lids (Collier[13]), so that one can be certain the fibers concerned in these movements lie in this neighborhood. Riley concluded that the superior colliculus was either a relay station in the suprasegmental pathway or a subcortical center, similar to the pontile center for lateral gaze. The impulses presumably get into the posterior longitudinal bundle but, unlike the impulses for lateral gaze, do not, according to Spiegel and Sommer, first run through the vestibular nuclei (fig. 5). They also expressed the opinion that these impulses may not even run into the posterior longitudinal bundle. They eventually reach the appropriate nuclei of the muscles which raise and lower the eyes and the eyelids.

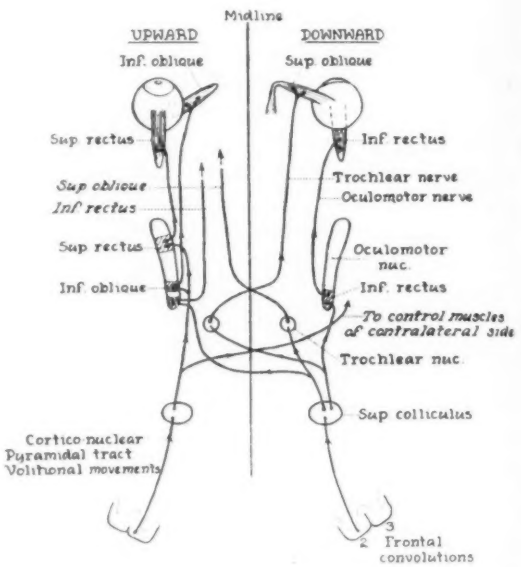

Fig. 5.—Diagram of the course of the fibers for vertical gaze. The left half of the diagram shows the course for upward gaze; the right half, that for downward gaze. According to Spiegel and Sommer, the fibers leave the pyramidal tract without entering the vestibular nuclei and run directly to the superior colliculi. From here they are distributed to the appropriate nuclei of the ocular muscles.

Centers and Pathways for Convergence.—The cortical center for the willed convergence movements of the eyes is unknown, unless it is the same frontal centers which produce conjugate deviation. There is plenty of evidence to support the belief that there is a subcortical center for convergence, for convergence alone may be paralyzed while the horizontal conjugate movements are retained and vice versa. There must be a special center for convergence, therefore. Brouwer placed this center in Perlia's nucleus, a single nucleus of small cells lying between the paired Edinger-Westphal nuclei, on the basis of his phylogenetic studies (fig. 6).

Centers and Pathways for Divergence.—There is less evidence of either a cortical or a subcortical center for willed movements in divergence, and this is to be expected, since there is no voluntary control of divergence (other than relaxa-

12. Adler, F. H.: Reciprocal Innervation of Extraocular Muscles, Arch. Ophth. **3**:318 (March) 1930.

13. Collier, J.: Nuclear Ophthalmoplegia with Especial Reference to Retraction of Lids and Ptosis and to Lesions of Posterior Commissure, Brain **50**:488 (Oct.) 1927.

tion of convergence) in the normal subject. Cases of bilateral willed divergence have never been recorded, and unilateral willed divergence may be brought about in rare cases by the relaxation on one side of the convergence impulse while it is maintained on the opposite side. Such cases do not prove the existence of a divergence center. Some authors, however, postulate the existence of such a center close to the sixth nerve nuclei (Bruce[14]). In the majority of cases of divergence paralysis the characteristic symptoms are soon followed by definite paralysis of one or both sixth cranial nerves, so that if a center exists it must lie close to these nuclei.

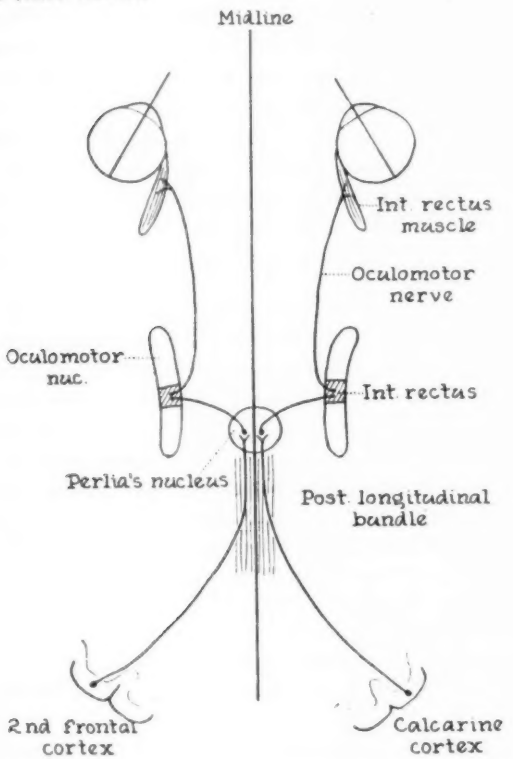

Fig. 6.—Diagram of the course of the fibers for convergence. A hypothetic fiber has been drawn from the second frontal convolution for voluntary convergence, although there is no experimental evidence to indicate the existence of this innervation. There is good evidence for the existence of the involuntary innervation from the occipital cortex, as shown on the right side of the drawing.

Although stimulation of the centers and pathways described in the preceding paragraph results in forced conjugate movements of the eyes, these parts of the central nervous system have to do normally only with voluntary, or willed, movements. If through disease or experimental destruction these centers are prevented from working, all the lower reflex movements of the eyes are still kept intact.

In man the importance of the reflex control of the ocular movements far outweighs that of the voluntary control. In nearly all the activities of the eyes the movements are dominated by the reflex control, although they may be initiated by a willed effort. The act of reading, for example, consists of a series of finely coordinated horizontal conjugate movements by means of which a certain span of words is fixed by the two maculas for a brief period of time. These shifts in fixation are carried out reflexly, in fact, entirely unconsciously, seven or eight being the usual number in an ordinary line of print. At the end of the line the eyes are moved to the left again by a voluntary effort of which the reader is aware and the visual axes moved down to the next line of print, when the process is repeated all over again. The act of reading would be a painfully slow process if these horizontal shifts had to be made voluntarily.

REFLEX CONTROL OF OCULAR MOVEMENTS

If there are gaps in knowledge concerning the willed ocular movements, knowledge regarding the reflex control of ocular muscle tone is still more meager, so that one is in no position today to get a complete picture of this important mechanism controlling the position of the eyes.

All willed movements of the body have to be coordinated and modified to fit in with changes in posture of the body as a whole or of its various parts; otherwise they become ataxic. The great coordinating center for smoothing out muscular activity in time and extent is the cerebellum. In the case of the eyes most of this is accomplished by impulses coming from the vestibular division of the eighth cranial nerve. In addition, there are other sources of tone which will be described, and perhaps many others the existence of which is not yet recognized.

A few examples will show how necessary such a mechanism is for assuring properly controlled movements of the eyes. When the head is in the erect posture, dextroversion of the two eyes in the horizontal plane is carried out mainly by the contraction of the right external rectus and left internal rectus muscles, together with reciprocal inhibition of their antagonists. If the head is tilted on the right shoulder, however, a contraction of these two muscles alone would not effect dextroversion in the plane of the horizon, but will turn the eyes obliquely down and to the right. Other muscles must therefore be brought into play to keep the eyes in the same plane of the horizon and to turn them to the

14. Bruce, G. M.: Ocular Divergence: Its Physiology and Pathology, Arch. Ophth. **13**:639 (April) 1935.

right. Hence the position of the head in space must modify profoundly the muscles which must be activated to produce any desired movement of the eyes. To this end the ocular muscles are kept under a constant changing state of tone through the vestibular nuclei by impulses coming in from the neck muscles and from the otolith apparatus in the labyrinths.

Further, when the head is tilted on the shoulder, the eyes are known to execute a torsional movement around their anteroposterior axes, so that as the head tilts the vertical meridians of the corneas stay erect and do not incline with the head. This compensatory torsional movement is brought about by an increase in tonus of those muscles which tend to keep the vertical meridians of the corneas erect, as the

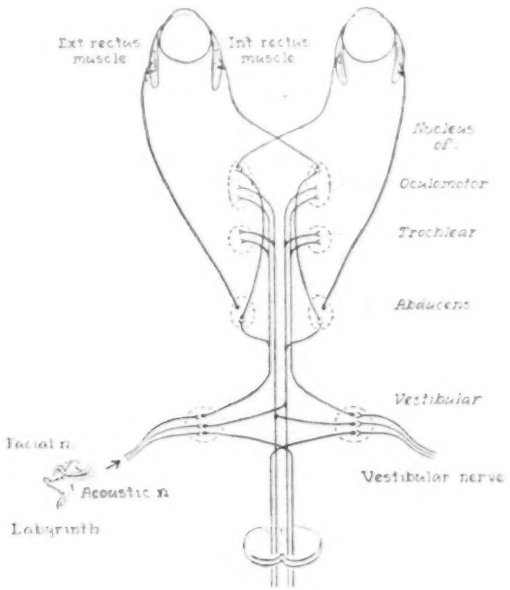

Fig. 7.—Diagram of the reflex innervation of the ocular muscles through the vestibular nuclei. From here the fibers enter the posterior longitudinal bundle and are then distributed to the ocular muscles. Tonic impulses from the neck muscles, the otolith apparatus and the semicircular canals are thus sent to the muscles.

head is tilted to the right or to the left. If the head is tilted to the right, for example, levotorsion must be produced in both eyes to keep the vertical meridians vertical. An increase in tone must therefore be sent to the right superior oblique and superior rectus muscles and to the left inferior oblique and inferior rectus muscles. These counterbalancing torsional movements are initiated by the otolith apparatus.

All of the sense organs of the body send impulses into the central nervous system which modify at one time or another the state of tone of the ocular muscles. A sudden loud noise causes the eyes to turn reflexly to the side from which it seems to come. A painful stimulus applied to the face causes the eyes to turn involuntarily to the place where it is located. Thus the cochlear division of the eighth nerve and the trigeminal nerve exert an influence on ocular muscle tonus, mediated through the posterior longitudinal bundle.

The most important reflex source of tone known, other than the visual, is that mediated by the vestibular division of the eighth nerve. Duke-Elder [15] has given a good description of the neuroanatomic details (fig. 7). This complex mechanism serves to correlate changes in posture and movements of the eyes by means of reflexes which, for the sake of study, may be divided into static reflexes, due to changes in position of the head in respect to gravity, and statokinetic reflexes, which occur as a result of movement of the head through space.

1. Static reflexes. These reflexes have already been mentioned briefly as altering the muscles called into play to secure any desired change of position of the eyes when the head is tilted on the neck. They arise as proprioceptive impulses from the neck muscles. As the head is tilted on the shoulders, the neck muscles of one side are put on a stretch, and this initiates the stretch reflexes, which are known to occur in most skeletal muscles. In the lower mammals the influence of the neck muscles is considerable, so that all the muscles of the eyes are under their tonic influence. In man their influence is felt much less, so that a rotation of the head on the trunk of 40 degrees produces a compensatory change in the position of the eyes of only 2 to 3 degrees (Fischer [16]).

Of more importance in man than the neck reflexes are the changes in tonus which arise as a result of changes in the position of the otolith organs of the labyrinths. All of the external ocular muscles, with the exception of the external and internal rectus muscles, are under the tonic control of the otolith apparatus, and changes in tilt of the head are compensated for by an effective change in tonus of the muscles which cause ocular torsion. It is the otolith apparatus which acts effectively to keep the vertical meridians of the corneas vertical when the head is tilted on the shoulder.

2. Statokinetic reflexes. Whenever the head is moved in space a movement of the fluid in the semicircular canals is set up which effects changes

15. Duke-Elder,[6] p. 292.

16. Fischer, M. H.: Messende Untersuchungen über die Gegenrollung der Augen und die Lokalisation der scheinbaren Vertikalen bei seitlicher Neigung (des Kopfes, des Stammes und des Gesamtkörpers): Neigungen bis 40°, Arch. f. Ophth. **118**:633, 1927.

in tone of some of the ocular muscles. The group of muscles affected depends on the semicircular canals which are stimulated, and this, in turn, depends on the posture of the head in respect to gravity during the movement in space. It is quite outside the field of this paper to elaborate on this subject, except to point out that such alterations of tone are constantly acting in the normal subject. The effect of such changes in tone is a conjugate movement of both eyes together laterally or in the vertical direction. It is customary to refer to the direction of nystagmus according to the quick component. If the quick component is to the right, one speaks of the nystagmus as being to the right. However, it is the slow component, in this case to the left, which is the essential part of nystagmus. The slow deviation of the eyes to the left is caused by tonic impulses from the labyrinths, and the quick return from this position is merely a phase of recovery. (The origin of this phase of nystagmus is still the subject of debate.)

Proprioceptive Sense of the Ocular Muscles.—A number of facts suggest the presence of a proprioceptive sense in the ocular muscles which orient and modify certain visual phenomena, such as projection and stereopsis. It is difficult to explain some of the phenomena of paralyses of ocular muscles without assuming some form of proprioception from the paretic muscle. On the other hand, most of the evidence refutes the existence of proprioception in its strictest sense.

In the general musculature of the body it is possible to prove true proprioception by demonstrating the presence of specialized end organs in the muscle, such as the muscle spindle, and by finding special fiber tracts in the central nervous system which carry these impulses set up in the muscle. The muscles with proprioceptive sense also have stretch reflexes, which can be proved physiologically.

In the case of the ocular muscles it has not been possible to demonstrate these various criteria. Irvine [17] found that the muscle spindles were much fewer and of more primitive type than those present in skeletal muscle, and he could not demonstrate the presence of any sensory nerves from the extraocular muscles and tendons. McCouch and Adler [18] were unable to detect any stretch reflexes in decerebrate cats, and Hoffmann [19] failed in the same way in human beings. There is certainly no anatomic or physiologic proof that proprioceptive sensation is mediated from the ocular muscles. According to Irvine and Ludvigh,[20] almost all the facts seem more compatible with the hypothesis of an innervation sense than with that of a proprioceptive sense. From this point of view, muscular activity still forms the basis for the judgment of direction when the eyes are moved, but this "judgment is not founded on the actual contraction of the muscles but upon the 'will' to move them." When the eyes are moved to the right in a dark room, one is conscious of the position they are in not from proprioceptive sensations from the muscles but from the willed effort in turning them.

In support of this hypothesis one can point to the fact that when the eyes are moved reflexly, the subject is quite unconscious of their position—in fact, often of their movement. In the case of nystagmus due to labyrinthine stimuli, whether resulting from disease or artificially induced by rotation in a chair or by douching the ear with cold water, the subject does not know that his eyes are moving but, instead, is conscious only of the apparent movement of the room. Everything around him seems to be turning, while he feels certain that neither his eyes nor the rest of his body is moving. There may, however, be a sensation of falling. The same is true of optokinetic nystagmus. During the movements of the eyes which occur on looking out of a moving railway coach at the passing scenery, the subject experiences no sensation that his eyes are undergoing constant nystagmus. Pulling the eye laterally with forceps gives no sense of its change in position.

Under no conditions in which the eyes are deviated by reflex sources or mechanical pull is the subject aware of that deviation—which he should be if there were proprioception from the ocular muscles themselves—and yet when the eyes are moved by a willed effort, he knows that they have moved and can tell exactly what position they occupy in respect to the orbit.

Penfield and Erickson [3] found that sensation of movement of the eyes, although a rare response, resulted from stimulation of the conscious subject in an area of the frontal cortex anterior to the fissure of Rolando in a zone near area 8 (fig. 8). The knowledge of the position of the eyes may be something akin to stereognosis. The elements which are essential for stereognosis are point localization, discrimination of two points and the sense of position and movement (muscle-tendon sense), and these in the case of the hand,

17. Irvine, S. R.: Histology of Extra-Ocular Muscles, Arch. Ophth. **15**:847 (May) 1936.

18. McCouch, G. P., and Adler, F. H.: Extraocular Reflexes, Am. J. Physiol. **100**:78 (March) 1932.

19. Hoffmann, P.: Ist es möglich, die physiologischen Erfahrungen über die Sehnenreflexe (Eigenreflexe) mit den pathologischen in Einklang zu bringen? Nervenarzt **2**:641 (Nov. 15) 1929.

20. Irvine, S. R., and Ludvigh, E. J.: Is Ocular Proprioceptive Sense Concerned in Vision? Arch. Ophth. **15**:1037 (June) 1936.

for example, are located in the cortex in the sensory motor region fairly close to the motor representation for the muscles of the hand. It cannot be said that this is evidence either for or against a true proprioceptive sense.

If a patient has recent paresis of an extraocular muscle, for example the right external rectus, he will past point at an object held in the right motor field, even when he uses the right eye only. There are two possible explanations for this past pointing. It may be assumed that in order to turn his paretic right eye out to fix the object he has to contract the muscle more than he normally would, and the proprioception from the muscle therefore gives rise to the sensation that the object is farther to the right than it really is—hence he points to the right, past the object.

On the other hand, Duke-Elder [21] pointed out that a paretic muscle fails to carry the eye out fully, so that the image falls not on the macula of this eye but on a point slightly to the nasal side of the macula. The fact that the right eye is not turned out far enough is not realized, for there is no real proprioception from the muscle, but only a gross knowledge of the willed effort made in turning the eyes. The image now falls on the nasal side of the macula and hence is projected out farther into the temporal field. Thinking that his eye has actually turned out the whole distance then causes him to past point to the position in space which has the local sign of the part of the retina stimulated.

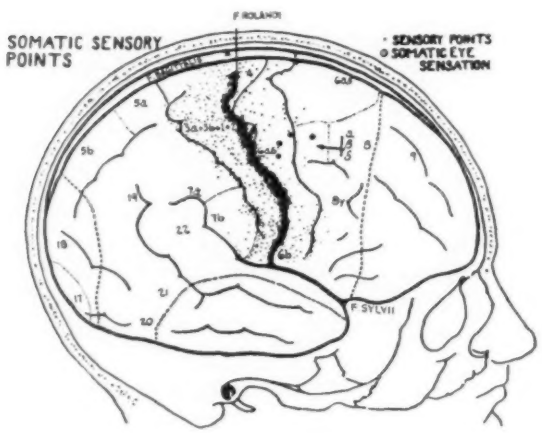

Fig. 8.—Diagram of the right motor cortex, showing the points where stimulation in the conscious subject produced a sensation of movement of the eyes. From Penfield and Erickson.[3]

In order to test which of these hypotheses is correct the following experiments were done.

A subject with recent paresis of the right external rectus muscle was seated at a Zeiss perimeter, and the left eye was covered. A test spot of light was then placed on the arc of the perimeter in the temporal field 30 degrees from fixation. A black cloth was held in such a way that the subject could not see his own arm while he was asked to point directly to the test spot of light. The difference between the actual position of the spot and the position to which he pointed was then measured. Two test objects were used: a large white spot of light measuring 15 mm. and a small spot of light measuring 2 mm. and illuminating a letter or small word. This word was small enough that the subject had to turn the eye all the way out so that its image fell on the fovea before he could read it.

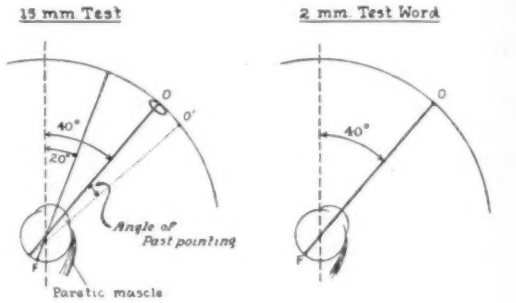

Fig. 9.—Right eye of a subject with paresis of the right external rectus muscle. On the arc of a Zeiss perimeter a light, O, has been placed at 40 degrees in the temporal field. With the left eye covered, the subject is requested to look at the object and to point with his right hand to it. The right hand is shielded from his view entirely, so that he is not conscious of where he is pointing. The angle of past pointing is then measured.

On the left the diagram shows that the eye does not turn out to 40 degrees to look at a large test object, i. e., a 15 mm. light, but turns out only to 20 degrees, this being sufficient for the subject to see the light. The image therefore falls on the nasal side of the fovea and hence is projected into space at O'. The subject therefore past points to O'.

On the right the diagram shows what happens when a small test light and word are used, of such a nature that in order to see what the word is the eye must be turned out all the way so that the image falls on the fovea. In this case the projection is correct and there is no past pointing.

It was found that when the large spot of light was used the angle of past pointing was much larger than when the small spot and letter were used. When the large spot is used, the subject does not have to turn his eye out all the way so that the image falls on the fovea in order to see it, and under these circumstances his past pointing is considerably greater than when he looks at the small spot, which must fall on the fovea (fig. 9).

Now, if the angle of past pointing depended on false proprioception from the palsied muscle, it would be either the same or greater when the

21. Duke-Elder,[3] p. 1052.

small spot was used, for the eye has to turn out farther for this test object than for the large test spot. The angle of past pointing is determined by the position of the image on the retina. When this falls on the fovea, as it does when the small spot is used, the projection is good. When the eye is not turned out quite far enough and the image falls on the nasal side of the fovea, as it does when the large test spot is used, the projection is to the right of the object to that part of space which has the local sign of the retinal receptors on which the image falls.

This experiment was done on 2 subjects with recent palsy of the external rectus muscle and on 1 normal subject in whom an injection of procaine hydrochloride along the external rectus muscle produced a paresis of this muscle, with typical limitation of movement and homonymous diplopia for five hours.

W. R., suspected of having a brain tumor, had paresis of the left external rectus muscle, of three weeks' duration. No other muscles were involved. Central visual acuity was normal. The fields were normal. There were choking of the disks of 1 D. and pronounced hypertensive changes in the retinas.

Position of Test Object, in Degrees	Angle of Past Pointing, in Degrees	
	15 Mm. Test Spot	2 Mm. Test Spot and Word
10	18	2
10	18	0
10	18	0

G. McC., had paresis of the right external rectus muscle of two weeks' duration. The condition followed spinal anesthesia for operation on the gallbladder. No other muscles were involved. The eyes were otherwise normal.

Position of Test Object, in Degrees	Angle of Past Pointing, in Degrees	
	15 Mm. Test Spot	2 Mm. Test Spot and Word
10	8	3
10	5	3
10	5	5

In F. H. A., a normal subject, paresis of the right external rectus muscle was produced by injection of 2 per cent procaine hydrochloride. The figures given in the tabulation were obtained after typical paresis was produced.

Position of Test Object, in Degrees	Angle of Past Pointing, in Degrees	
	15 Mm. Test Spot	2 Mm. Test Spot and Word
10	5	0
30	3	2
40	6	3

In the third case measurements taken before the injection of the procaine showed no difference in the angle of projection (there was no past pointing) when the large test spot and the small spot and light were used.

The Visual Fixation Reflex.—The most important source of reflex activity in man comes from the visual impulses themselves. The fovea, being the point of greatest visual acuity, must be placed so that the image of an object in space falls on it in order to be distinctly seen. The voluntary movements of the eyes take care of this when there is a conscious need for foveal vision. But, in addition, nature has further provided that once the image is on the macula it will stay fixed there, reflexly, or if it is near the macula, the eye will be moved reflexly so that the fovea receives the image. This result is attained by an involuntary reflex, called variously the visual fixation reflex, the fusion reflex (when both eyes are so moved that the image falls on each macula), the optomotor reaction, etc. Whenever the image of a moving object tends to move off the macula in one direction, an increase in tonus occurs in those muscles which move the eyes in that direction; thus the eyes are reflexly turned to follow the object (figs. 10, 11 and 12).

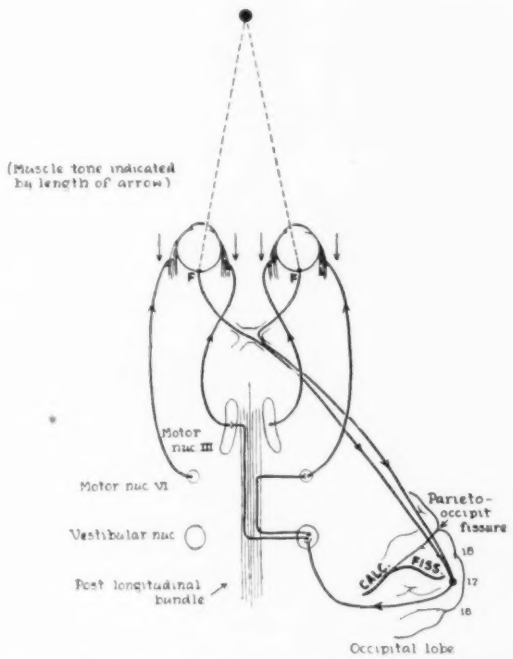

Fig. 10.—The image of the dot straight ahead of the eyes falls on the foveas. The impulses are carried up to the extreme tip of the occipital lobe along the calcarine fissure. The optomotor fibers carry impulses from here to the vestibular nuclei, which distribute tone to the ocular muscles (amount of tone indicated by length of arrow in diagram). As long as the images are on the foveas, the tone of the muscles turning the eyes to the right is equal to that of the muscles turning the eyes to the left. The diagram purposely begs the question of the bilateral representation of the foveas. It may be imagined that the same stimuli go to the left calcarine cortex, as is shown here to the right.

This reflex is demonstrated in daily life by watching a person looking out of a railway coach at the surrounding scenery as it passes by the window. The eyes will be found to make a series of movements consisting of a slow phase in the direction the objects seem to be moving and a

rapid phase in the opposite direction. The movements continue as long as the person has his eyes open and pays attention to the scenery. The person may or may not be aware of the fact that his eyes are moving. This is the so-called railroad nystagmus. It can be simulated in the laboratory by having the patient look at black and white stripes on a moving drum. Here it is called optokinetic nystagmus.

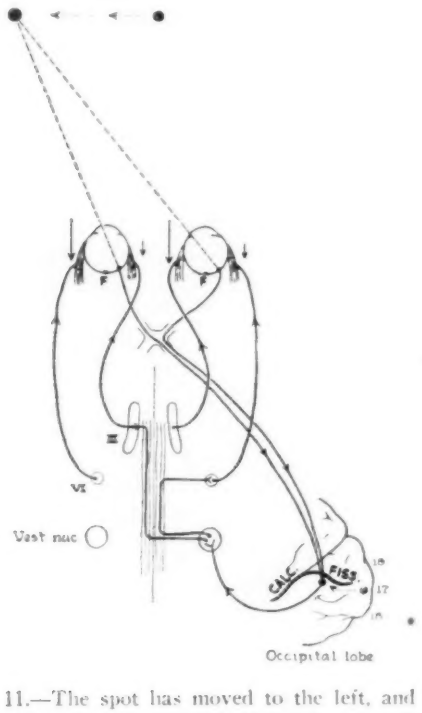

Fig. 11.—The spot has moved to the left, and therefore its image has moved off the foveas onto the right halves of the two retinas. The visual impulses are carried up to the occipital cortex, to cells which lie farther forward along the calcarine fissure than those which come from the foveas. From these cells an optomotor impulse is sent to the vestibular nuclei which diminishes the tone of the muscles which turn the eyes to the right and increases the tone of those which turn the eyes to the left. The eyes are therefore turned to the left until the image of the spot once more falls on the foveas.

The same reflex produces involuntary fusional movements of each eye in an effort to maintain binocular single vision when weak prisms are held up in front of one eye while the subject looks at a light at 6 meters. If a weak prism is held up suddenly base-out in front of the right eye, the right eye will quickly and involuntarily turn in to bring the fovea in line again with the object of regard. If the prism is weak, i. e., 1 to 2 prism diopters, the movement will be made without the patient's awareness. If the prism is stronger, or if more prism is continuously added, the patient will be conscious of a momentary diplopia, as the eye involuntarily makes the effort at fusion. There will come a time finally when the amount of prism added will be too great for the involuntary fusion reflex to overcome. The strength of the prism which breaks up the involuntary fixation reflex is the measure of the reflex fusional capacity.

Beyond this point it is still possible to add more prism base out and secure fusion by a voluntary effort. More and more prism may be added and the patient, after some practice, still finds that he can fuse the two images into one. When the point is reached where this is no longer possible, the strength of the prism used is the measure of the adducting power, or prism adduction. Since the patient soon learns that he can increase his convergence power by calling on his accommodation, he quickly learns to converge and accommodate as though he were looking at a near object. This presents him with a dilemma. He can fuse the

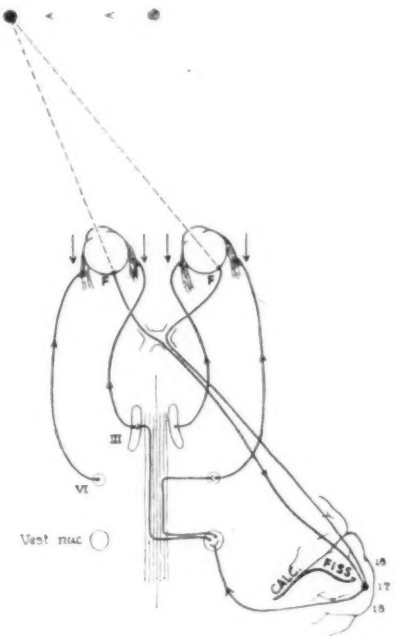

Fig. 12.—The eyes have been turned to the left by means of the visual fixation reflex to follow the movement of the spot. The images which now fall on the foveas again send their impulses up to the foveal localization of vision in the occipital cortex, and the optomotor impulses once again equalize the tone of the muscles turning the eyes to the right and of those turning the eyes to the left, so that the eyes remain fixed as long as the image of the spot remains stationary.

distant double images of the light by converging and accommodating, but in using his accommodation he obtains blurred images. The trick then, he soon finds out, is to keep together, i. e., to fuse, two images into a single blurred image. That this has been called the adduction power, and thought of by some as representing the strength of a subject's muscles of convergence, is testimony to the naiveté of the examiner. After prisms are prescribed for home use, the patient returns with marked improvement in his ability to overcome prisms base out. He has, therefore, according to such an unwitting physician increased the power of his internal rectus muscles, when all that he has done is to learn to dissociate convergence from accommodation, or to accept fused blurred images, rather than double clear ones.

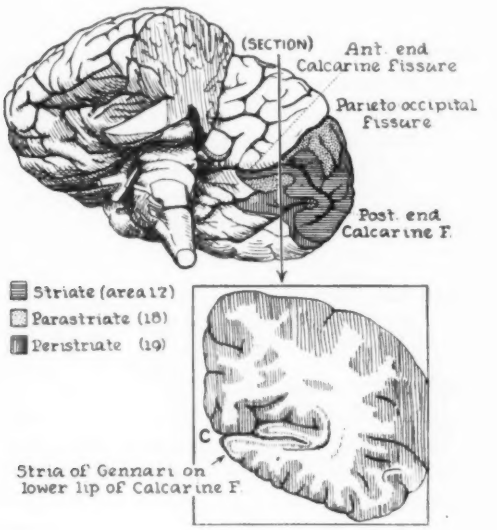

Fig. 13.—Diagram of the mesial half of the right occipital cortex, the left occipital lobe having been cut off. The insert shows a transverse section through the calcarine fissure at the level of the arrow. At this level the parieto-occipital fissure joins the calcarine fissure, so that only the lower lip of the calcarine fissure has the white line of Gennari. Some investigators believe that the cells giving rise to the optomotor impulses of the visual fixation reflex arise in the striate area (area 17 of Brodmann), but according to Hines [23] they originate in the cells of areas 18 and 19 of Brodmann.

The visual fixation reflex is composed of an afferent and an efferent pathway. The afferent pathway [22] is the visual pathway, running from the cones of the fovea through the lateral geniculate body and ending in the cells of the calcarine cortex (fig. 13). There is no general agreement, however, in which layer of cells of the visual cortex the motor fibers begin. Most authors consider the cells of area 17 of Brodmann as the beginning of the efferent arc, but Hines [23] gave evidence for believing that the optomotor pathway begins in the cells of layer 18, the parastriate area, and layer 19, the peristriate area, rather than in the striate area itself, or layer 17.

From this optomotor center fibers run into the posterior longitudinal bundle. Their exact pathway is unknown, but Spiegel and Sommer [24] have traced them as follows:

Starting from the optomotor zone surrounding the area striata, the fibers seem to pass under the angular gyrus and are observed in "the lateral wall of the posterior and of the inferior horn of the lateral ventricle, internal to the corticopetal optic fibers, in the internal sagittal stratum. Farther on they are found partly in the pretectal area and in the roof of the anterior corpora quadrigemina and in the lateral geniculate body; some fibers could also be traced by the degenera-

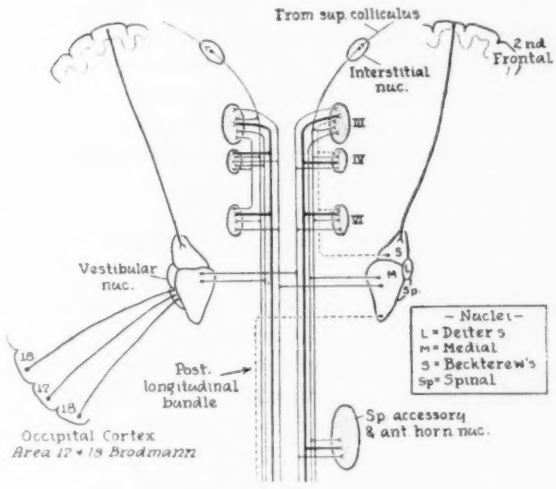

Fig. 14.—Diagram of the posterior longitudinal bundle, showing the voluntary motor innervation of the ocular muscles on the right side and the reflex innervation from the occipital lobes on the left side. It is not intended to indicate that the fibers end in one or another of the vestibular nuclei, as the exact course of these fibers is unknown. The drawing shows a fiber ending in a certain nucleus, e. g., Deiters', merely for convenience.

22. An excellent review of the central visual pathway is that of Kronfeld, P. C.: Central Visual Pathway, Arch. Ophth. **2**:709 (Dec.) 1929.

23. Hines, M.: Recent Contributions to Localization of Vision in Central Nervous System, Arch. Ophth. **28**:913 (Nov.) 1942.

24. Spiegel and Sommer,[9] pp. 391-392.

tion method to the basis pedunculi, reaching the pontine nuclei (Barris). . . . From the rhombencephalon on, the course of the oculogyric tract seems to be common with that for the vestibular reflexes to the eyes; in other words the corticofugal impulses apparently use the path of the vestibulo-ocular reflex to reach the eye muscles [fig. 14]." The impulses reach the appropriate ocular muscles through the posterior longitudinal bundle.

Ter Braak [25] divided optokinetic nystagmus into two kinds: The first requires the factor of attention, and the second seems to be independent of the frontal cortical centers and, in fact, may be elicited in animals after extirpation of the cerebral hemispheres; this he refers to as the subcortical, or passive, type.

Accommodation Reflex.—Reference has already been made to the effect of an abnormal amount of accommodation, resulting in excessive convergence and thus giving rise to accommodative convergent squint. There is developed normally a close reflex association between accommodation and convergence; this link is not rigid, but somewhat elastic. By orthoptic training it is possible to make this linkage less rigid, so that a hyperopic person may retain the necessary accommodation to focus on distant objects, yet relax the amount of convergence that would ordinarily be associated with it and thus retain parallelism of his visual axes.

ORTHOPHORIA AND HETEROPHORIA

The anatomic basis for the voluntary and reflex control of ocular movements has been outlined in so far as it is known at the present time. In the normal adult this mechanism keeps the two eyes associated with one another in all directions of gaze, so that the two foveas receive the images of the object of regard. The ocular muscles are under many sources of tone, which are acting constantly and varying among themselves according to the needs of the moment. Even when the eyes are in the primary position and are not being moved, they are kept there by a constant source of tone to the appropriate muscles. This position must not be thought of as one of rest, therefore.

The position of absolute rest is that assumed when the eyes are freed from every source of tone, a condition which occurs only with very deep anesthesia, coma or death. Under these conditions the eyes assume the anatomic position of rest, which is divergence of each eye about 10 degrees, and usually sursumvergence of about 5 degrees. During the waking hours the eyes are never in a position of rest.

It is possible to eliminate some of the sources of tone to the ocular muscles and to determine the position the eyes assume under these conditions. Only the visual sources of tonus can be eliminated readily. If, for example, the subject relaxes his attention and, as one says, lets his thoughts wander from the objects surrounding him, the position of the eyes may change. In some people they diverge greatly. If the image of one eye is made poorer than that of the other by placing smoked glasses of increasing density in front of it, a point will be reached where binocular vision is no longer possible, and the eyes, freed from the fusional impulses of the fixation reflex, will take up that position dictated by the remaining sources of tone, whatever they may be.

Whenever the vision of one eye interferes with the use of the two eyes together, an attempt is made by the body to free the eyes from this embarrassment, and the new position which the eyes assume will be that which best eliminates the confusion. This new position may not be the same as that assumed when the eyes are free of the fusional impulses, and therefore dictated by the other, remaining sources of tone, as previously described, but may be a forced position due to new sources of tone from the occipital cortex for the purpose of getting rid of diplopia. The body finds that when the two images cannot be fused together, they can be separated by deviating the eyes from one another, and that this separation helps to avoid the confusion, for one of the images may now be suppressed more easily and so the visual axes are turned either in or out to separate the images.

It has become customary to measure the position of the two eyes when the visual fusional impulses are either totally or partially removed and to term this the patient's phoria. If, under these conditions, the visual axes remain so that the image of the object of regard still falls on the two foveas, orthophoria is said to be present. On the other hand, if the images do not fall on the two foveas, heterophoria is said to be present, and the character and degree of the deviation are then determined and measured. It is unfortunate that the implication has arisen that orthophoria is the normal condition and heterophoria an abnormal one, for this is not true. By such standards there are no normal people. It is true that some people are orthophoric for distance when measured by the usual test of the Maddox rod, or even the cover test, but these are few, the

25. ter Braak, J. W. G.: Untersuchungen über optokinetischen Nystagmus, Arch. néerl. de physiol. **21**: 309, 1936.

majority showing 1 or 2 degrees of esophoria or exophoria. When the Maddox rod test is applied, or when the cover test is used, however, almost no one will show orthophoria for near vision. If the Maddox rod test for near vision is conducted in a lighted room, particularly if the patient is asked to measure his own deviation on a scale, such as the Thorington test for near vision, there may be orthophoria, due to the stimulus of accommodation, but the examiner has retained under these conditions a part of the visual fusional stimuli he originally sought to eliminate.

The various methods used to dissociate the two eyes will give different measurements, the amounts depending on the extent to which they eliminate the visual fusional stimuli. Heterophoria determined with the Maddox rod tells the effectiveness of dissimilar retinal images in holding the eyes straight as compared with the effectiveness of similar images. (The test should be performed in a dark room, so that there is no influence from the surroundings.)

Heterophoria determined with the cover test tells how effective monocular retinal stimulation is as compared with stimulation of the two retinas simultaneously. Vertical prisms used to determine the lateral phorias show what the eyes will do when they are presented with artificially produced double images which, although similar, cannot easily be fused; the results here are surely not the measure of a fusion-free position, but are most likely that of some forced position which the eyes assume in order best to avoid the confusion of the double images; measurements taken with this method are usually smaller than those with either the Maddox rod or the cover test.

Unfortunately, one does not know exactly what all the other sources of tone are which, combined, give the new position of the eyes freed from all visual fusional impulses. Some of them have already been enumerated, but it is likely that there are others as yet unknown. At present none of them can be eliminated experimentally in human beings.

Two conditions have been studied recently which may throw light on these involuntary mechanisms. These are the effects of alcohol and of anoxemia.

It is commonly supposed that the diplopia which, unfortunately, occurs when alcohol is taken to excess is due to the release of the eyes from visual fusional impulses, similar to the benumbing of all the senses. Any heterophoria which was present before the alcohol was taken would then become manifest; if a person had an esophoria before he drank, he would have an esotropia while under the influence of alcohol, and if he had an exophoria before drinking, he would have an exotropia afterward. This has been shown to be incorrect by a number of investigators.

Powell [26] demonstrated that a definite tendency toward esophoria always developed in a series of of men and women when given alcohol. This change was constant, gradual and progressive. It was recovered from in about ten hours, and the muscle balance returned to its previous level. It made no difference whether the person was esophoric or exophoric before the alcohol was given; the change was always in the direction of esophoria. The muscle balance for near vision changed in the direction of progressive exophoria. The angle of convergence (convergence near point) showed a progressive decrease, while the prism divergence was not determined.

Powell explained the progressive esophoria at 6 meters on the basis of a weakening of the external rectus muscles and, in defense of this, called attention to the fact that the abducens nerve is termed the weakling of the cranial contents. He explained the decrease in convergence power on the basis of diminished tone and control of the internal rectus muscles.

In 1940 Colson [27] repeated these experiments by giving 21 normal adults 2 ounces (39 cc.) of rye or scotch whisky every half-hour and testing their muscle balance and voluntary ductions for distance. He found a gradually increasing esophoria in every case, which in 2 instances reached the stage of strabismus with diplopia. The minimum change was 2 prism diopters; the maximum was 11 prism diopters. It made no difference what the original muscle balance was before the alcohol was taken.

Duction tests showed that according to the usual notation the abduction was reduced in every case, but if allowance was made for the state of the muscle balance, there was no change in the abduction. The real adduction was decreased in every case, but in the figures the author gives this decrease is not striking. Both the conventional and the real adduction were hardly changed at all in 2 cases and were only greatly reduced in 1 case, and these were the only ones in which measurements were made. From this observation one could hardly draw any conclusions regarding duction power.

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26. Powell, W. H., Jr.: Ocular Manifestations of Alcohol and Consideration of Individual Variations in Seven Cases Studied, J. Aviation Med. **9**:97 (June) 1938.

27. Colson, Z. W.: Effect of Alcohol on Vision, J. A. M. A. **115**:1525 (Nov. 2) 1940.

From these experiments it cannot be determined how alcohol produces esophoria for distance. One might guess that it produces its effect by stimulation of the convergence center. The production of torsional movements in cats under the influence of barbiturates has already been mentioned. The mechanism may, of course, be the release of inhibition from higher centers on the convergence center, so that too great an innervation to the internal rectus muscles is set free. It may be a weakening of the divergence mechanism. Further pharmacologic investigation is needed before it can be ascertained where alcohol acts.

The eyes act in a similar way under the influence of severe anoxemia. Although Wilmer and Berens [28] found that anoxia produced in a low pressure chamber resulted in an increase of any preexisting heterophoria, Velhagen [29] reported the same kind of changes as those which occur in acute alcoholism, i. e., a pronounced development of esophoria. No matter what the subject's muscle balance was to start with, according to this author, the change is always in the direction of esophoria as the atmospheric pressure is reduced. At 3,000 meters his results were not conclusive, but at 5,000 to 6,000 meters the increase in esophoria was considerable. At the near point there was a change similar to that with alcohol, i. e., a tendency toward increasing exophoria. Velhagen spoke of this as a convergence insufficiency. Curiously, he suggested that the changes he observed were probably due to alterations in the muscles themselves, as a result of changed metabolism due to the anoxemia, since he was unable to satisfy himself that there was any increase in convergence tone or hyperexcitability of the convergence center.

I have repeated these experiments on the effects of anoxia and have been able to verify and further to amplify Velhagen's results. These investigations will be reported on later in the ARCHIVES. It was found that during oxygen lack all subjects become more esophoric whether they were esophoric or exophoric to start with. There was no constant change in their muscle balance for near vision. During oxygen lack there was a shift in the range of fusion toward the convergent position, but the range itself showed little variation from normal. This would indicate that during anoxia the convergence center is either released from inhibition or is directly stimulated.

28. Wilmer, W. H., and Berens, C., Jr.: The Effect of Altitude on Ocular Functions, J. A. M. A. **71**:1394 (Oct. 26) 1918.

29. Velhagen, K., Jr.: Heterophorie unter den Bedingungen des Höhenfluges, Luftfahrtmedizin **1**:344, 1937.

COMMENT

The following should be considered possible sources of abnormal convergence tone:

Proprioception from the Ocular Muscles.—If true proprioceptive impulses originated in the ocular muscles, as in other skeletal muscles, a theory of strabismus might be conceived on the basis of abnormal proprioception. All the evidence points to a different mechanism, however, and one is forced to the conclusion that the body obtains information of the position of the eyes in respect to the orbits from the pathways of willed innervation, and not from the muscles directly.

There are grounds for believing that the position of the eyes in the orbits influences the sensory response, however. It has frequently been noted that patients who have divergent strabismus with equal vision in the two eyes and who ordinarily alternate cannot be made to see binocularly through one of the major stereoscopes even when the tubes are placed at the angle of squint. As soon as the eyes are straightened by operation, however, they see binocularly; some have first or second degree fusion, and still others stereopsis. A plausible explanation would seem to be that binocular vision is possible only when the eyes are in the natural position in respect to each other in the orbits, and that when they are divergent, owing to faulty innervation of the ocular muscles, binocular vision is inhibited—even though the images are presented to the two eyes along the visual line of each.

Cortical Centers for Convergence.—If there are cortical centers for voluntary convergence, which there probably are, their location is unknown at present. The occipital centers for the visual fixation reflex must give rise to convergence tone in keeping the foveas fixed on an object either approaching or receding from the eyes. There is as yet no evidence which could incriminate this mechanism. I have examined a number of children with strabismus to see whether their optokinetic nystagmus differed from that of normal children, and the results so far are negative. The movements have not been photographed as yet, however, and no conclusions can be drawn. Other methods of investigating the fusional fixation reflex should be devised, as this is an attractive possibility to be explored.

Subcortical Centers and Pathways for Convergence and Divergence.—Under conditions of acute intoxication with alcohol or as a result of severe anoxia, the visual axes become convergent and an esophoria frequently becomes an esotropia. Unfortunately, it is not yet known what causes this—whether there is a stimulation of the convergence center, a release of this center from

cortical inhibition or a paralysis of the divergence mechanism. It is likely that either the first or the second possibility is the true cause. If the change were due to a paralysis of divergence, why is it that during sleep or anesthesia the position is that of divergence? It seems more probable that the convergent position is due to hyperactive tonic impulses sent to the internal rectus muscles.

There is too little evidence for one to do more than suggest that the type of convergent squint under discussion might be caused by a similar mechanism in the growing infant. But it is not improbable. There can be no doubt that comitant squint is due to an abnormal supranuclear source of tonus, for by its very nature the angle of squint remains the same in all positions of gaze. (For an excellent discussion of comitance see Chavasse.[2]) It can be readily seen that in the infant if the eyes were forced into the convergent position from a hyperexcitable convergence center, the result would be just what it is—one eye would take up fixation instead of both eyes remaining convergent, while the other eye would assume the whole of the angle of convergence. The result would be a fixing eye and a squinting eye; and if the two eyes were of equal value (same refractive error and acuity of vision), the squint would be an alternating one. When one eye took up fixation, the whole burden of the overconvergence would be on the fellow eye. If one eye were better than the other (greater refractive error or some other obstacle to vision), then it would remain the fixing eye, and a monocular squint would develop.

Vestibular Apparatus.—Impulses reaching the posterior longitudinal bundle through the vestibular nuclei are of considerable importance in regulating the tone of the ocular muscles. Vestibular impulses cause conjugate movements of the eyes laterally and vertically, and in man these movements are always equal. Bartels[30] found in rabbits that stimulation of one labyrinth affected the tone of the muscles of the ipsilateral eye more than of the opposite eye, but these results do not apply to man. Besides, it has never been shown that vestibular tone has anything to do with convergence. This seems to be the only associated movement of the eyes which is not affected by changes in vestibular tone, and one can hardly imagine, therefore, that squint is produced by an abnormality of this mechanism.

Sommer[31] examined a large series of normal persons, patients with high esophorias and patients with convergent squint and found no effect of caloric stimulation of the vestibular apparatus on the phoria or the angle of squint. In cases of so-called oblique strabismus, in which the squinting eye turned upward on adduction and downward on abduction, Ohm[32] claimed to have demonstrated a vestibular component, but the evidence presented is not convincing.

CONCLUSIONS

In all cases of convergent squint, except those in which the condition is due to paralysis of an ocular muscle, the fundamental cause is an abnormal convergence innervation. There is no difference between an esophoria and an esotropia save that in the former the eyes are still held together by the visual fusional stimuli whereas in the latter this mechanism has failed to maintain fusion. Regardless of the state of perfection of fusion, the important factor in the causation of squint is the force which produces it, and that is an excessive convergence innervation.

In the case of accommodative squints one does not have far to look for the origin of the excessive convergence, and Donders' theory for this condition is as correct today as it was when he formulated it. There are many squints, however, which are not of this origin, and in these one must look elsewhere for the source of, and the reason for, the excessive convergence. From a review of the physiologic mechanisms concerned in normal ocular movements, one can find possible sites of interference which could lead to comitant squint, but as yet the evidence is merely suggestive. It is sufficient, however, to warrant further research, free from the bias of any preconceived theory.

Hospital of the University of Pennsylvania.

30. Bartels, M.: I. Ueber Regulierung der Augenstellung durch den Ohrapparat, Arch. f. Ophth. **76**:1, 1910; II. Schielen und Ohrapparat, ibid. **77**:531, 1910; III. Kurven des Spannungszustandes einzelner Augenmusklen durch Ohrreflexe, ibid. **78**:129, 1911; IV. Die stärkere Wirkung eines Ohrapparates auf das benachbarte Auge, ibid. **80**:207, 1911.

31. Sommer, I.: Labyrinth und Schielen, Ztschr. f. Augenh. **65**:14 (May) 1928.

32. Ohm, J.: Schrägschielen, Arch. f. Augenh. **99**: 619 (Nov.) 1928.

USE OF METHYL CELLULOSE IN OPHTHALMOLOGY

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There has been need in ophthalmology for a nonirritating and chemically inert colloid which would dissolve in water to produce a viscous, colorless solution having a high degree of transparency and a refractive index similar to that of the cornea. Such a solution would be useful as a bland vehicle for ophthalmic medicaments, as a substitute for natural secretions in cases of keratoconjunctivitis sicca and as an emollient and cohesive solution to be used with contact lenses and gonioscopic prisms. Attempts have been made to adapt various compounds for these purposes, with only partial success. Acacia, tragacanth and gelatin have been most widely used, but they are chemically unstable, have high refractive indexes and are good mediums for the growth of bacteria and fungi. A synthetic substance, methyl cellulose,[1] has considerable advantages over these naturally occurring gums. Its properties and some of its ophthalmic uses are reported here.

Methyl cellulose is a water-soluble cellulose ether with a high degree of purity and uniformity. It is produced in six types, graded according to the degree of viscosity. For ophthalmic purposes the highest viscosity type (4,000 centipoises) is preferable, as only a small quantity need be added to aqueous solutions to increase the viscosity greatly without significantly altering its optical properties. Two per cent aqueous solutions have a viscosity many times that of water, but are highly transparent and colorless and have a low dispersion power and a refractive index (1.336) similar to that of distilled water (1.334). In addition, solutions of methyl cellulose are neutral, odorless, tasteless and nearly inert chemically. They are stable in the p_H range which is tolerated by the eye and seem unaffected by light or aging. Boiling produces coagulation, but on cooling, methyl cellulose redissolves. Therefore solutions may be sterilized by boiling. Aqueous solutions may be diluted with ethyl alcohol and are compatible with the drugs commonly used in ophthalmology. Solutions containing only pure methyl cellulose do not support bacterial or fungal growth.

It is recommended that solutions of methyl cellulose be prepared by first mixing the dry material thoroughly with approximately half the required volume of water at boiling temperature and allowing it to soak from twenty to thirty minutes, with agitation. This procedure hastens wetting of the methyl cellulose. The solution does not become clear until the remaining amount of water is added and the mixture cooled to room temperature. Solutions of maximum clarity may be obtained by reducing the temperature to 5 or 10 C. during the cooling period and stirring until smooth. Undissolved fibers may be removed by centrifugation.

When used as a vehicle for ophthalmic drugs, a 0.5 to 1 per cent solution of the 4,000 centipoise type of methyl cellulose may be instilled with a small bore dropper, but higher concentrations are more satisfactorily administered in collapsible tubes with small tips. The emollient properties of the colloid reduce irritation of the conjunctiva produced by drugs. Unlike the commonly used oily vehicles of high viscosity, methyl cellulose solutions mix well with secretions and spread smoothly throughout the conjunctival sac. Also, vehicles containing methyl cellulose remain longer in the conjunctival sac than simple aqueous solutions and therefore result in more effective absorption of many ophthalmic drugs; however, highly surface-active compounds, such as tetracaine and dibutoline sulfate (dibutylcarbamate of dimethylethyl-β-hydroxyethyl ammonium sulfate) are adsorbed by the colloid, so that their penetration of the tissues is retarded. Methyl cellulose is best suited to form stable, nonoily suspensions of slightly water-soluble substances, such as sulfamerazine. For example, 1 to 4 Gm. of finely powdered sulfamerazine stirred into 2 per cent solution of methyl cellulose in distilled water forms a nonirritating and highly effective unguentum for local treatment of external inflammations of the eye. The drug has a higher affinity for the tissues than for this vehicle; consequently, therapeutically effective concentrations in the con-

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This investigation is part of a study being conducted under a grant from the John and Mary R. Markle Foundation.

1. The preparation used was methocel (Dow Chemical Company).

junctiva and cornea are readily obtained. Chemical analyses[2] of normal conjunctiva excised from 3 volunteers one-half hour after a single instillation of 3 per cent sulfamerazine in a 2 per cent aqueous solution of methyl cellulose revealed an average concentration of sulfamerazine of 42 mg. per hundred grams in the specimens from the lower fornix and 18 mg. per hundred grams in the specimens from the extreme upper fornix.

Solutions of methyl cellulose have cohesive and emollient properties which, along with a neutral reaction, a high degree of clarity and a refractive index similar to that of the cornea, make them superior to simple aqueous solutions for use with contact lenses. Methyl cellulose solutions may be buffered to the alkaline levels usually desired for use with contact lenses, but the first 3 patients to use the solution have found buffering unnecessary. The viscous methyl cellulose solution adheres to the lens and therefore permits the contact lens to be applied to the eye with much less chance of the solution spilling and being replaced by air bubbles than if a simple aqueous solution is used. Also, methyl cellulose solutions have a lubricating action similar to normal conjunctival mucus, so that patients are able to wear the lenses more comfortably and for longer periods when methyl cellulose is added to the aqueous solution. A concentration of 0.5 to 1 per cent of the 4,000 centipoise type of methyl cellulose dissolved in 0.9 to 1.2 per cent sodium chloride is most satisfactory for use with contact lenses. Bubbles accidentally stirred into solutions containing up to 1 per cent methyl cellulose will rise to the surface but will be retained in solutions of higher viscosity.

Methyl cellulose solutions increase the practicability of contact lenses applied for gonioscopy or for examination of the fundus with the corneal curvature neutralized. These solutions are particularly suitable for use with gonioscopic lenses, such as the type recently developed by Allen.[3] In this type of lens the water chamber is eliminated and the lens is held in apposition with the cornea by capillary attraction. A highly cohesive film is obtained by moistening the surface of the lens before application with isotonic solution of sodium chloride containing 1 per cent methyl cellulose. The lubricating and emollient properties of the colloid permit prolonged application of the contact lens without damage to the delicate corneal epithelium. The Allen gonioscopic lenses have been made of a plastic material with the same refractive index as the cornea and the methyl cellulose solutions, so that apparently perfect optical continuity is obtained.

A 0.5 per cent solution of the 4,000 centipoise type of methyl cellulose containing 0.9 to 1.25 per cent sodium chloride has provided an excellent substitute for deficient conjunctival secretions in 2 patients with keratoconjunctivitis sicca. In both patients a superficial punctate disturbance was evident on biomicroscopic examination with the slit lamp, and Schirmer's test was positive before treatment was undertaken. The corneas became clear, and subjective symptoms were relieved by instillations of the methyl cellulose solution at two to three hour intervals. Buffering of the solution was unnecessary. The only complaints referable to use of the colloid were that immediately after instillation it produced an unpleasant sensation of the lids being stuck together. Also, excesses of the colloid would dry on the lashes, but the fine fiber could be removed easily with a damp cloth.

No cases of hypersensitivity to methyl cellulose were noted among the approximately 100 patients who received instillations of this colloid. Also, Hueper, Martin and Thompson[4] were unable to produce hypersensitivity to the colloid with repeated intravenous injections in dogs.

Deichmann and Witherup[5] found that the orally ingested methyl cellulose could be completely recovered in the feces, and Hare and Clark[6] observed that the colloid was not absorbed from closed subcutaneous wounds. Likewise, methyl cellulose did not appear to be absorbed from the conjunctival sac. Weighed quantities of the colloid sealed into the conjunctival sac of rabbits by suturing of the lids and closing of the lacrimal passages were completely recovered after several hours.

Instillations of 1 per cent solution of methyl cellulose into the conjunctival sac of rabbits were made two to three times daily for three months. No evidence of injury to the ocular tissue was found by either biomicroscopic examination with the slit lamp or histologic methods. Practically no inflammatory reaction resulted from the subconjunctival injection of 0.1 cc. of a 1 per cent solution of methyl cellulose in albino rabbits,

2. Sulfamerazine—Chemical Reagent, Brochure P. H. 689, Philadelphia, Sharp & Dohme, Inc., 1943.

3. Allen, L.: A New Contact Lens for Viewing the Angle of the Anterior Chamber of the Eye, Science **99**:186 (March 3) 1944.

4. Hueper, W. C.; Martin, G. J., and Thompson, M. R.: Methyl Cellulose Solution as a Plasma Substitute, Am. J. Surg. **56**:629 (June) 1942.

5. Deichmann, W., and Witherup, S.: Observations on the Ingestion of Methyl Cellulose and Ethyl Cellulose by Rats, J. Lab. & Clin. Med. **28**:1725 (Nov.) 1943.

6. Hare, R., and Clark, E. N.: Use of Plastic Vehicles for Applying Sulfonamide Compounds to Wounds, War Med. **4**:140 (Aug.) 1943.

but similar injections into the anterior chamber produced mild iridocyclitis. This finding is in accordance with the observation of Hare and Clark [6] that in closed wounds methyl cellulose creates a foreign body reaction. It would appear, then, that the use of methyl cellulose in the conjunctival sac is a safe procedure except in instances of open wounds leading into the eyeball.

SUMMARY

Methyl cellulose is a synthetic colloid with a high degree of purity and uniformity. In cold water, it forms colorless, stable, neutral, viscous solutions with a high degree of clarity, low dispersive power and a refractive index similar to that of the cornea. In the conjunctival sac these solutions mix well with the conjunctival secretions and have an emollient action. Preliminary clinical investigations indicate that methyl cellulose is superior to naturally occurring gums or oils as a vehicle for many ophthalmic drugs, as a substitute for normal secretions in cases of keratoconjunctivitis sicca and as an emollient and cohesive medium for use with contact lenses and gonioscopic prisms.

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OPHTHALMIC PRISMS

CORRECTIVE AND METRIC

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CORRECTIVE PRISMS

Seventy-five years ago, in 1870, lenses were still being prescribed in terms of their radiuses of curvature (a description) rather than in terms of their power (an operational specification). Twenty years later the metric operational system of numbering lenses according to the work they did (in refracting light rays) was almost universal, and this proved to be so much more satisfactory that progressive ophthalmologists began to turn their attention to similar problems with prisms, which were labeled in terms of the apex angle, a unit of shape rather than a unit of strength.

In 1887 Jackson [1] examined the prisms in seven trial cases, representing four well known manufactures; he found that "in only one set did they all come within 16 per cent of the proper standard," and recommended that prisms should be marked according to their angle of minimal deviation. Landolt approved this recommendation, and a committee (Landolt, Burnett and Jackson) was appointed to present the proposition to the Tenth International Medical Congress, to be held in Berlin in 1890.

Before this meeting was held, however, Dennett [2] proposed to the American Ophthalmological Society a new unit, the centrad (∇), an operational unit specifying that in the position in which only one surface contributes to the deviation, the deviation shall be measured in hundredths of a radian. The centrad was thus a power measurement of the arc of the deviation produced. At the annual meeting of the American Ophthalmological Society in 1890, this unit, the centrad, was officially accepted.[3]

In the meantime Charles Prentice, a New York optician, had devised yet another operational metric unit, the prism diopter (Δ), a description of which was published in the *Archives of Ophthalmology* in January 1890.[4] The prism diopter is an operational unit; that is, it specifies work done, it is simple to understand and it fits into the metric system with a pleasing analogy to the dioptric system in specifying lens strengths. The prism diopter specifies that in the position in which only one surface contributes to the deviation, the deviation shall be measured on a tangent scale at a distance of 1 meter, each centimeter of deviation representing 1 prism diopter (1Δ). This unit has grown in usage and importance until at present it is practically universally employed (at least in America) for specifying and calculating the prismatic effects to be incorporated into ophthalmic corrective lenses.

At the Tenth International Medical Congress, held in Berlin in 1890, the Landolt, Burnett and Jackson report was discussed, but no action was taken.[5]

In the years 1889 to 1892 considerable acrimonious discussion took place, chiefly between the advocates of the prism diopter and those of the centrad. Owing to the lucid and satisfactory analyses of the problems encountered in calculating and incorporating prism effects in corrective lenses and to the development of an accurate prismometer and the use of prismatic scales, the prism diopter of Prentice gradually gained the ascendancy, which it has held to this day.

Since these discussions practically always revolved about the use of corrective, or therapeutic, prisms (as opposed to metric, or measuring, prisms of higher power), an important fact was usually overlooked. In order to attain the simplicity of either the centrad (∇) or the prism diopter (Δ) an unusual condition must be intro-

From the Knapp Memorial Laboratories, Institute of Ophthalmology.

1. Jackson, E.: The Designation of Prisms by the Angular Deviation They Cause, Instead of by the Refracting Angle, Tr. Internat. M. Cong. (Sect. 11) **3**:785, 1887.

2. Dennett, W. S.: A New Method of Numbering Prisms, Tr. Am. Ophth. Soc. **5**:422, 1888-1890.

3. Tr. Am. Ophth. Soc. **5**:467, 1888-1890.

4. Prentice, C. F.: A Metric System of Numbering and Measuring Prisms, Arch. Ophth. **19**:64 and 128, 1890.

5. Tr. Internat. M. Cong. **4**:49, 1890.

duced into the method of measurement. This condition, imposed both by Dennett and by Prentice, requires that *all refractive effect must take place at one surface.* Stated in this manner, the implication is rather startling, but this is the equivalent of specifying, as they both did, that the angle of incidence on the first surface must be zero; i. e., the prism is to be held with its first surface normal to the ray which is to be refracted. Thus all effect must take place at the second surface, and, as in the vertex specification of ophthalmic lenses, this gives a definite and easily located plane from which to make measurements.

Thus, four methods of numbering prisms are available.

1. The dimensions of the apex angle (α), expressed in degrees ($^\circ$).

2. The actual deviation, in degrees, produced by the prism when it is set at its position of minimal deviation, i. e., when the ray within the prism is perpendicular to the line bisecting the apex angle (δ° min).

3. The centrad (∇), or arc centune. This is an angular measurement of the deviation, equaling, as the name indicates, one hundredth of a radian. On the arc of a circle of 1 meter radius each centimeter of deviation would equal 1 centrad. The incident ray must be normal to the first surface.

4. The prism diopter (Δ), or tangent centune. This expresses a linear, or tangential, measurement. With the incident ray perpendicular to the first surface, the prism diopter notation expresses the number of centimeters of displacement produced by the prism as measured on a tangent plane situated 1 meter from the second surface.

As has been seen, the apex angle (α) does not tell what deviation will be produced unless one knows the refractive index and the position in which the prism is held.

The minimal deviation (δ°min) is the most exact, scientific and satisfactory specification, since it precisely expresses the angular deviation produced at an exact and symmetric position. It is the specification universally used by physicists. From the ophthalmologic viewpoint it has the disadvantage that the angle of incidence of entering light continuously varies with the apex angle, and from this viewpoint some fixed position of holding is desirable.

The centrad has no particular advantage over the prism diopter. It expresses an exact deviation, but only when the position of holding is known.

For all practical uses, in specifying prisms to be incorporated with lens prescriptions (up to 8Δ) the departure from the specification that the incident ray is to be normal to the surface is unimportant, and the advantages accruing from simple and easy calculation leave the Prentice system far in advance of any other method of notation.

METRIC PRISMS

With metric prisms (prisms used for measuring high degrees of deviation, as in strabismometry) the situation is quite different, and the Prentice method offers many pitfalls and serious dangers. An extensive series of measurements and calculations of prisms to be used for this purpose is being undertaken and will be reported by one of us later. In justice to Prentice, it should be noted that the faults found with this system should not be attributed to him (he was concerned chiefly with corrective prisms, for which his system serves admirably). The dangers arise from the uncritical use of a system under conditions in which its faults are most glaringly manifest.

Our present purpose is to note the conditions on which the Prentice system is based and to report measurements begun thirteen years ago by one of us (L.H.H.) and recently carried to completion by the group.

In 1890 prisms were not commonly used to measure strabismus. Most squints were estimated by the Hirschberg method, or the deviation was measured on the perimeter. The latter method is still the choice of most European and of many American ophthalmologists. Not until twenty years later, largely under the influence of Duane, was the prism and cover test popularized. This method is now widely taught and used, particularly in America.

As we have noted, in 1890 and for a long time thereafter, most prisms were numbered according to the degrees ($^\circ$) encompassed by their refracting angle (α). Many prisms still in use are so marked, and manufacturers are not uniform in their notations. No set of standards has been universally employed, and few if any manufacturers indicate in their markings the unit used. Some manufacturers continue to use the apex angle as a unit for marking; others label prisms in terms of minimal deviation in degrees, and still others use the prism diopter, almost all, however, failing to indicate the unit used.

As a result prisms bearing the same label vary widely in their powers of refraction, and the owner of a set is liable to be misled in interpreting the results of his measurements.

In 1931 one of us (L.H.H.) conducted measurements on eleven sets of prisms belonging

to himself, his friends and various ophthalmic clinics. The results showed wide variations. Last year further elaborations for measurements were set up and the data extended to include twenty sets of prisms, two of which were sets of plastic prisms.

METHOD

Light from a 6 ampere ribbon filament (*A*, fig. 1) was collimated, passed through a mask (*B*), a narrow vertical slit (*C*), a 73 Eastman Wratten monochromatic filter (*D*) to a condensing lens (*E*), positioned to collimate rays proceeding from the slit. The central beam was selected by a mask over the first surface of the condensing lens, and over the lower half of the second surface was placed a covering which carried an accurate line positioning the optical axis of the beam. At the other end of the optical bench was placed a transit head gonioscope, which gave vernier readings to 1 minute. This was supplied with two accurately ground positioning plates, so that the location of the second face of the prism was exact and its distance from the receiving surface was precisely 1 meter. Elaborate measurements were taken to insure the correct prism to screen measurement, and the correct (90 degree) angle between the optical axes and the screen surface. The receiving surface was a frosted white glass. The measurement of tangent deviations were accurate to ± 0.5 mm.

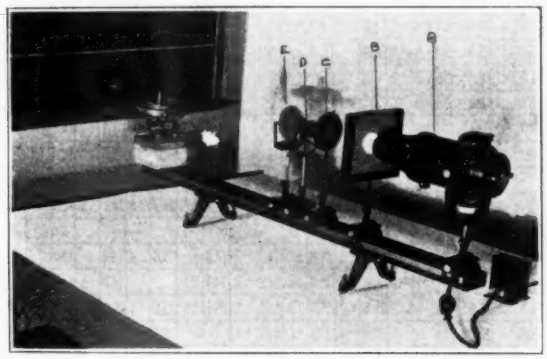

Fig. 1.—Apparatus used in checking and measuring prisms.

All measurements were made as tangent values and checked against goniometric readings.

Measurements were made of the apex angle (α) of each prism, of its deviation in the position of minimal deviation, of its deviation in the Prentice position, and of its deviation in two other positions, to be discussed later (the "split positions" and the two positions of maximal deviation, when possible). Frequent checks were made by positioning the prism for a right or a left hand reading (reversing apex for base and first surface for second surface). The apex angle (α) was checked by reflecting the slit beam back along its course first from one surface and then from the other surface, and a final check on the accuracy of the measurements was made by calculating the index of refraction from the formula involving apex angle and deviation. A variation of more than 0.004 of the index required rechecking of all measurements of a given prism.

RESULTS

The results are shown in figure 2.

The column marked Δ gives the power of the prism as measured in the Prentice position, and the column marked *E* gives the error expressed in prism diopters (Δ).

It is immediately apparent that the numbers on some sets of these prisms (sets 1, 2, 3, 4, 5, 12 and 20) refer to apex angles rather than to prism diopters. If this were not true, they would be in error over 100 per cent in some cases. For example, the prism marked 40 in set 1 actually has a power of 81Δ in the Prentice position, and this corresponds roughly with its apex angle (40° 10′), while the prism marked 50 could not be measured in the Prentice position since its apex angle (50° 2′) was greater than the critical angle of glass (41° 2′)—when this prism was used in the Prentice position, total internal reflection took place. The same is true of sets 2, 3, 4, 5, 12 and 20.

Of the prisms apparently marked in prism diopters, the set of plastic prisms (no. 6) was slightly strong in the lower powers (greatest error, 12 per cent for 8Δ) and slightly weak in the higher powers (about 10 per cent too weak for 40Δ, 45Δ and 50Δ). Errors among the glass prisms were not distributed regularly and bore no relation to strength except that, as might be expected, the higher powers tended to show greater total errors.

Reasonable manufacturing tolerances for ophthalmic measuring prisms would be as follows:

	Allowance
Up to 5Δ	0.06Δ
5Δ to 9Δ inclusive	0.12Δ
10Δ to 12Δ inclusive	0.25Δ
14Δ, 16Δ and 18Δ inclusive	0.37Δ
20Δ, 30Δ and 40Δ inclusive	0.50Δ

Most of the prisms we measured did not meet these tolerances, especially in the higher powers.

Most prism sets we examined did not have a manufacturer's label to indicate their source. None of them had an identifying unit to indicate the significance of the numeric label.

The importance of this lack is obvious and is well illustrated by the two sets of prisms 11 and 12. These two sets came from the office of a well trained and careful ophthalmologist. If they were properly used, a squint with a deviation of 40Δ would be exactly neutralized by the 40 prism in set 11; but if set 12 should be chosen, a squint with a deviation of 78Δ would be neutralized with the 40 prism.

These results show only the errors found when we were measuring prisms under rigid, specified conditions. Many other measurements were made in checking the apex angle (α), the minimal deviation produced by each prism (δ° min)

and the two maximal deviations when possible (δ° max, i=90° and δ° max, e=90°), as well as the deviation produced in a newly specified position ($\delta\Delta$ split). A tremendous effect can be produced on the deviation caused by a prism by departing from the conditions specified, i. e., by permitting the angle of incidence to deviate from zero. These measurements confirmed by calculated values will be reported by one of us later.

SUMMARY

The prism diopter (Δ) is a unit invented and introduced by Prentice, an optician, to denote the operational power of ophthalmic prisms.

artificial condition, namely, that the surface of incidence shall be normal to the path of the entering ray. While this condition has no practical significance for prisms of the powers used in corrective lenses, it has a high degree of significance for strong metric prisms used in measuring strabismus.

Some sets of prisms used by ophthalmologists are calibrated in prism diopters (Δ); others are calibrated in terms of the apex angle, in degrees (°). The unit of calibration is rarely given. This results in possible errors, due to misconception of the unit used, of over 100 per cent in the higher powers. In the lower powers such errors are insignificant for practical clinical

SET	1		2		3		4		5		6		7		8		9		10		11		12		13		14		15		16		17		18		19		20	
Δ	Δ	E	Δ	E	Δ	E	Δ	E	Δ	E	Δ	E	Δ	E	Δ	E	Δ	E	Δ	E	Δ	E	Δ	E	Δ	E	Δ	E	Δ	E	Δ	E	Δ	E	Δ	E	Δ	E	Δ	E
.5	.4	-.1	.5				.6	+.1			.6	+.1	.4	-.1	.5		.4	-.1	.6	+.1	.4	-.1							.5		.4	-.1	.5				.5			
1	1.0		1.0		1.0		1.1	+.1	.9	-.1	1.0		.9	-.1	1.0		1.1	+.1			.9	-.1	1.0				1.0		1.0		1.1	+.1	1.0		1.0		1.0		1.0	
2	1.7	-.3	1.9	-.1	2.0		2.1	+.1	2.0		2.0		2.0		2.1	+.1	2.0		1.9	-.1	2.0		1.9	-.1			1.9	-.1	2.1	+.1	2.0		2.0		1.9	-.1	2.0		2.0	
3	2.9	-.1	3.0		3.0		3.0		2.9	-.1			3.0		2.8	-.2	3.0				3.0		3.0		2.9	-.1	3.0		2.8	-.2	3.0		3.0		3.0		3.0		3.0	
4	4.0		4.0		3.9	-.1	4.0		3.9	-.1	4.1	+.1	4.0		4.1	+.1	4.1	+.1	4.0		4.0		3.5	-.5	4.0		3.9	-.1	4.1	+.1	4.1	+.1	4.0		3.9	-.1	4.0		3.9	-.1
5	5.0		5.1	+.1	5.0		5.1	+.1	5.0				5.0		4.9	-.1	5.0		4.9	-.1			4.4	-.6	4.8	-.2	5.0		4.7	-.3	5.0		4.9	-.1	5.0		4.7	-.3	4.9	-.1
6	6.0		5.8	-.2	5.8	-.2	5.6	-.4	5.8	-.2	6.1	+.1	6.0		6.0		5.9	-.1	5.8	-.2	6.0		5.9	-.1	5.8	-.2	6.0		6.0		5.9	-.1	5.8	-.2	5.9	-.1	5.8	-.2	5.1	-.9
7	7.0		6.9	-.1	7.0		6.9	-.1	7.0				6.8	-.2	6.9	-.1	7.0		7.0		6.9	-.1	6.1	-.9	6.8	-.2	6.8	-.2	6.9	-.1	7.0				6.8	-.2			6.1	-.9
8	7.8	-.2	7.8	-.2	8.0		7.9	-.1	8.4	+.4	9.0	+1.0	8.0		7.8	-.2	7.9	-.1	7.7	-.3	7.9	-.1	7.3	-.7	7.8	-.2	7.8	-.2	7.8	-.2	7.9	-.1	7.9	-.1	7.8	-.2	7.9	-.1	7.0	
9	8.0	-1.0	9.0		8.9	-.1	9.1	+.1	9.0				9.0		8.9	-.1	8.9	-.1	8.9	-.1	9.0		8.2	-.8	9.0		8.8	-.2	8.9	-.1	8.9	-.1	9.1	+.1	8.8	-.2	9.1	+.1	8.1	-.9
10	10.0		9.8	-.2	10.1	+.1	9.8	-.2	9.8	-.2	10.2	+.2	9.8	-.2	9.7	-.3	10.0		9.9	-.1	10.0		9.6	-.2	9.5	-.5	9.8	-.2	9.7	-.3	10.0		9.8	-.2	9.8	-.2	9.8	-.2	10.4	+.4
11	11.0		11.0				11.8	+.8					10.9	-.1	10.7	-.3	10.6	-.4	10.9	-.1									10.7	-.3	10.6	-.4	10.8	-.2			10.8	-.2		
12	12.0		11.8	-.2	11.8	-.2	11.8	-.2	10.8	-1.2	12.5	+.5	11.8	-.2	11.8	-.2	11.7	-.3	11.8	-.2	12.0		11.9	-.1	11.7	-.3	11.7	-.3	11.9	-.1	11.7	-.3	11.7	-.3	11.7	-.3	11.7	-.3	12.1	+.1
13	13.1	+.1	12.9	-.1			13.1	+.1					12.8	-.2	12.8	-.2			12.8	-.2									12.8	-.2										
14	13.0		13.9	-.1	12.9	-1.1	12.8	-1.2	13.8	-.2			13.8	-.2	13.8	-.2	13.6	-.4	13.7	-.3	14.0		13.0	-1.0			13.7	-.3	13.8	-.2	13.7	-.3	14.0		13.6	-.4	14.0		13.0	-1.0
15	14.9	-.1	15.0				14.9	-.1			15.2	+.2	14.7	-.3	14.8	-.2	14.8	-.2	14.8	-.2					14.7	-.3			14.8	-.2	14.8	-.2	14.7	-.3			14.7	-.3		
16	15.8	-.2	16.0		15.8	-.2	16.2	+.2	14.5	-1.5			15.7	-.3	15.7	-.3	15.8	-.2	15.6	-.4	15.9	-.1	15.4	-.6			15.6	-.4	15.7	-.3	15.8	-.2	15.5	-.5	15.6	-.4	15.6	-.4	15.9	-.1
18	18.2	+.2	17.6	-.4	17.4	-.6	17.9	-.1	18.0				18.0		17.4	-.6	17.3	-.7	17.4	-.6	18.0		17.8	-.2			17.6	-.4	17.4	-.6	17.4	-.6			17.6	-.4			17.9	-.1
20	19.9	-.1	19.8	-.2	20.1	+.1	19.8	-.2	20.0		20.2	+.2	19.5	-.5	19.7	-.3	19.2	-.8	19.4	-.6	20.0		20.1	+.1	19.7	-.3	17.6	-.4	19.7	-.3	19.3	-.7	19.5	-.5	17.9	-2.1	19.6	-.4	20.2	+.2
25	26.9	+1.9	27.1	+2.1			27.5	+2.5	27.9	+2.9	25.7	+.7	27.3	+2.3	24.2	-.8	24.3	-.7	24.3	-.7					24.7	-.3			24.2	-.8	24.3	-1.7	24.6	-.4			24.6	-.4		
30	36.6	+6.6	30.3	+.3	37.1	+7.1	36.7	+6.7	36.5	+6.5	27.3	-2.7	36.4	+6.4	29.1	-.9	29.3	-.7	29.1	-.9	30.2	+.2	36.8	+6.8	29.1	-.9	29.4	-.6	29.1	-.9	29.9	-.1	29.5	-.5	27.9	-.6	29.5	-.5	38.5	+8.5
35											33.1	-1.9			33.5	-1.5	33.1	-1.9							31.8	-3.2			33.6	-1.4	33.1	-1.9	34.7	-.3			34.7	-.3		
40	81.0	+41.0	60.2	+20.2	76.3	+36.3	76.9	+36.9	74.3	+34.3	36.3	-3.7	39.9	-.1	37.8	-2.2	37.0	-3.0	37.3	-2.7	40.0		78.3	+38.3	36.0	-4.0	38.2	-1.8	37.8	-2.2	37.0	-3.0	38.6	-1.4	38.1	-1.9	38.5	-1.5	50.7	+10.7
45											40.8	-4.2													41.8	-3.2														
50											45.8	-4.2	43.4	-6.6	46.2	-3.8	46.7	-3.3	48.1	-1.9					51.3	+1.3			46.2	-3.8	46.8	-3.2	49.8	-.2			49.7	-.3		

Fig. 2.—Results of measurements on twenty sets of prisms. The first column at the left indicates the number borne by the prism—presumably, prism diopters (Δ). Under each set number the left hand column, marked Δ, indicates the measured power of the prism, and the right hand column, marked *E*, indicates the error, expressed as + (too strong) or — (too weak).

It was intended to be used mainly for corrective prisms, and in this respect the concept has great value, particularly in facilitating the work of the optician who is required to incorporate prismatic effects into a prescription for lenses. By extension, the unit was utilized to express the strength of all prisms up to a theoretic limit of 115Δ (or up to that prism whose apex angle coincides with the critical angle of the medium used).

To achieve this simplification, Prentice was forced to incorporate into his system a highly artificial condition, namely, that the surface of incidence shall be normal to the path of the entering ray. While this condition has no practical significance for prisms of the powers used in corrective lenses, it has a high degree of significance for strong metric prisms used in measuring strabismus.

Some sets of prisms used by ophthalmologists are calibrated in prism diopters (Δ); others are calibrated in terms of the apex angle, in degrees (°). The unit of calibration is rarely given. This results in possible errors, due to misconception of the unit used, of over 100 per cent in the higher powers. In the lower powers such errors are insignificant for practical purposes. The purchaser of a set of prisms is entitled to know the unit used, and this should be clearly marked on each prism.

Measurements made on twenty assorted sets of prisms indicated that many of them have errors exceeding reasonable manufacturing standards.

In use of prisms labeled with the Prentice system great care must be observed to comply with the conditions on which this system is based; otherwise high errors will be introduced by inaccurate positioning.

OCULAR SEQUELAE OF ADMINISTRATION OF GENERAL ANESTHESIA

PARALYSIS OF THE SUPERIOR RECTUS MUSCLE, EXOPHTHALMOS, PSEUDOPTOSIS AND FIBROSIS OF THE INFERIOR RECTUS MUSCLE FOLLOWING AN OPERATION FOR PILONIDAL CYST WITH THE PATIENT UNDER GENERAL ANESTHESIA

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AND
CAPTAIN GERALD FONDA
MEDICAL CORPS, ARMY OF THE UNITED STATES

A search of the literature revealed only one report of paralysis of the extraocular muscles associated with general anesthesia [1] other than spinal anesthesia. In Woltman's report 2 cases are described. However, there have been approximately 175 cases of paresis of the extraocular muscles related to the use of spinal anesthesia [2] and 1 case in which this condition was associated with local anesthesia administered for tonsillectomy.[3]

In the case to be described, paralysis of the right superior rectus muscle, pseudoptosis of the right upper eyelid, exophthalmos of the right eyeball and fibrosis of the right inferior rectus muscle were diagnosed after administration of ethylene-ether anesthesia for an operation for pilonidal cyst.

REPORT OF A CASE

F. W., a patrolman aged 36, was operated on for pilonidal cyst on Jan. 29, 1942, under ethylene-ether anesthesia. Prior to this operation the patient's eyes revealed no abnormality.

Within twenty-four hours after the operation the patient experienced diplopia when looking upward. On the following day diplopia was present while he was reading. The onset of ptosis was coincidental with the diplopia.

On Feb. 18, 1942, three weeks after the operation for pilonidal cyst, examination of the eyes revealed that vision without correction was 20/20 in each eye; the near point of accommodation for the right eye was 180 mm. for 400 mm. print, and the near point for the left eye, 180 mm. for 300 mm. print. The reading of the exophthalmometer, set at 97 mm., was 19 mm. for the right eye and 14 mm. for the left eye. Protrusion of the right eye was straight forward and completely reducible on pressure, and there was no retrobulbar tenderness or tumor of the eyeball. The right cornea was hypersensitive to touch. The levator muscle was not paralyzed, but the depression of the eyeball made elevation of the upper eyelid difficult. The pupils were equal in size and reacted promptly to light and in accommodation. The media and the fundi were normal. The right eye could be elevated to within 5 degrees of the primary position with slight extorsion. There was no action of the right superior rectus muscle. Abduction was excellent, but adduction was slightly limited. The eyelids opened and closed normally. The patient wrinkled his forehead normally. Sensitivity of the skin and of the conjunctiva was normal.

Physical examination revealed no abnormalities. The blood pressure was 140 systolic and 86 diastolic. The pulse rate was 80. Neurologic examination gave negative results except for the findings referable to the eye. No bruit was heard.

All the laboratory tests made within six weeks after the operation gave normal results. Lumbar puncture, a colloidal gold test and a Wassermann test revealed nothing significant. An electroencephalogram was normal. The urine was normal except for an occasional leukocyte. The basal metabolic rate was +6, +12 and +1, respectively, on three occasions. The blood cholesterol content was 240 mg. per hundred cubic centimeters. The blood iodine level was normal (8 micrograms per hundred cubic centimeters). Two roentgenograms of the skull, including the orbits, taken in February and November 1942, revealed no abnormality or pathologic change. The optic foramens were normal in size and shape. A roentgenogram of the right orbit taken after injection of air into the perior bital tissues showed the eyeball to be normal in size and position, with no evidence of tumor. The visual fields were within normal limits.

From the Research Department of the New York Eye and Ear Infirmary.

This study was aided by a grant from The Ophthalmological Foundation, Inc.

Presented before the Section of Ophthalmology of the New York Academy of Medicine, May 15, 1944.

1. Woltman, H. W.: Post-Operative Neurological Complications, Wisconsin M. J. **35**:427, 1936.

2. Levine, J.: Paralysis of Extra-Ocular Muscle After Spinal Anesthesia, Arch. Ophth. **4**:516 (Oct.) 1930. Fawcett, K. R.: Extra-Ocular Muscle Paralysis Following Spinal Anesthesia, Minnesota Med. **14**:648, 1931. Biggam, J.: Paralysis of Ocular Muscles Following Spinal Anesthesia (with Stovaine), Brit. J. Ophth. **16**:552, 1932. Blatt, N.: Ocular Paralysis After Spinal Anesthesia, Wien. klin. Wchnschr. **41**: 1048, 1928.

3. Kiraly, J.: Paralysis After Anesthesia of Tonsils for Tonsillectomy, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **138**:119, 1934.

On May 4, 1942 the right eye could be elevated upward only to 5 degrees below the primary position with slight extorsion.

Examination one year later revealed that there was essentially no change in the condition of the eyes. Examination of the eyes preoperatively on Dec. 7, 1943, eleven months after the onset, revealed the following status: The upward limit of fixation of the right eye had remained at 5 degrees below the horizontal plane. The eyeball, even with forceps, could be rotated upward only to the horizontal plane. The muscle balance, determined with the cover and parallax test, was 15 prism diopters of exotropia and 78 prism diopters of left hypertropia at 6 meters, and 22 prism diopters of exotropia and 78 prism diopters of left hypertropia at 25 cm. The findings in the diagnostic positions of gaze at 75 cm., with the cover test and prisms, were charted as follows:

	Right		Left	
Eyes:	Right and up	LHT 90Δ plus XT variable	LHT 90Δ XT 10Δ	Eyes: Left and up
	Right	LHT 80Δ	LHT 78Δ XT 12Δ	Left
	Right and down	LHT 78Δ	LHT 64Δ XT 12Δ	Left and down

In this tabulation LHT and XT indicate left hypertropia and exotropia respectively.

Study of the motor anomaly revealed complete loss of elevation in the field of activity of the right superior rectus muscle and severe secondary deviation of the left inferior oblique muscle. The elevating power of the right inferior oblique muscle could not be determined accurately, although the slight elevating power that was present seemed to be produced by that muscle. The right inferior rectus muscle showed considerable contracture. There was also severe secondary deviation of the left superior oblique muscle.

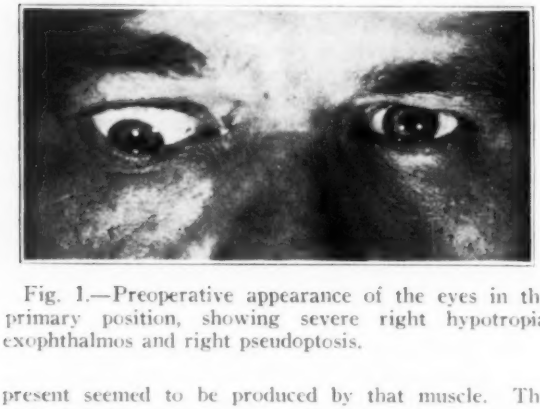

Fig. 1.—Preoperative appearance of the eyes in the primary position, showing severe right hypotropia, exophthalmos and right pseudoptosis.

The preoperative diagnosis was paralysis of the superior rectus muscle, exophthalmos, right pseudoptosis, partial paralysis of the inferior oblique muscle and fibrosis of the inferior rectus muscle of the right eye.

On Feb. 9, 1943, the following surgical procedure was performed: complete tenotomy of the right inferior rectus muscle at the insertion, resection of 8 mm. of the right superior rectus muscle and transplantation of the inferior half of the right lateral rectus muscle to the temporal half of the stump of the inferior rectus muscle (fig. 2). The upper half of the lateral rectus muscle was reattached to the lower half of the insertion of the lateral rectus muscle. There were no adhesions about the inferior rectus muscle, but there was absolutely no elasticity of this muscle when upward traction was produced with a tenotomy hook. It seemed to be completely fibrosed.

On March 1, 1943, three weeks following the operation, with the cover and parallax test the patient had orthophoria for distance (6 meters) and exophoria of 10 prism diopters for near (25 cm.) vision (fig. 3). The findings in the diagnostic positions of gaze with the cover test and prisms at 75 cm. were charted as follows:

	Right		Left	
Eyes:	Right and up	XT 12Δ LH 10Δ	XT 20Δ LH 9Δ	Eyes: Left and up
	Right	XT 8Δ RH 3Δ	XT 20Δ LH 5Δ	Left
	Right and down	XT 8Δ RH 16Δ	XT 16Δ LH 3Δ	Left and down

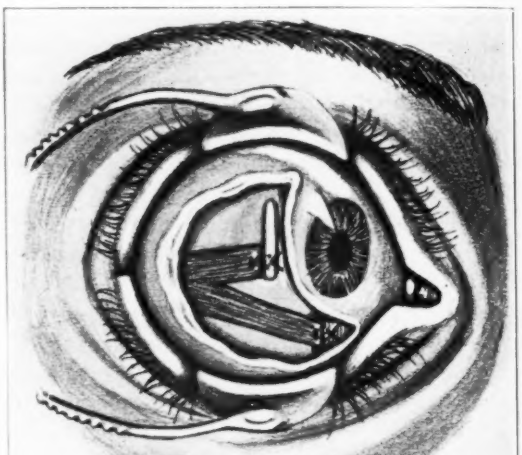

Fig. 2.—Illustration showing transplantation of the inferior half of the right lateral rectus muscle to the temporal half of the stump of the inferior rectus muscle and reattachment of the upper half of the lateral rectus muscle to the lower half of the lateral rectus insertion.

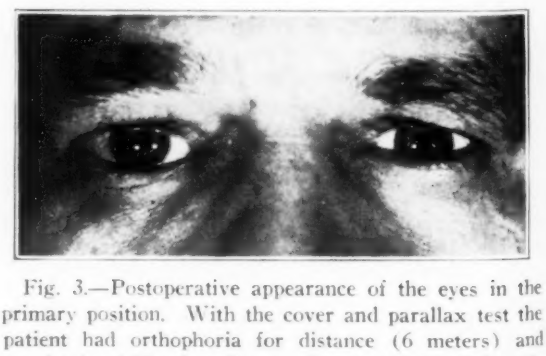

Fig. 3.—Postoperative appearance of the eyes in the primary position. With the cover and parallax test the patient had orthophoria for distance (6 meters) and exophoria of 10 prism diopters for near vision (25 cm.).

Three weeks after the operation, the patient obtained fusion, according to the Worth four dot test, at 6 meters and at 25 cm. The patient's near point of convergence was 80 mm. Slight weakness of the right superior rectus muscle and secondary overaction of the left inferior oblique muscle persisted. Underaction on depression in the field of activity of the right inferior rectus muscle continued. However, considerable improvement in ability to depress the right eye was noticed after several weeks. The right inferior oblique muscle recovered almost all of its normal power of elevation. The patient complained of diplopia when he looked upward, upward and inward, and upward and outward; he obtained fusion in other parts of the field (fig. 4). The lower eyelid was still displaced somewhat downward.

In January 1944, eleven months after the operation on the ocular muscles, the exophthalmos of the right eye seemed to have increased. This was verified by three different exophthalmometric readings taken during January and February 1944. The average of the three readings, with the exophthalmometer set at 98 mm., was 22 mm. for the right eye and 16 mm. for the left eye. The exophthalmometric reading taken in February 1942, one year prior to the operation on the eye, with the instrument set at 97 mm., was 19 mm. for the right eye and 14 mm. for the left eye. Since the patient has no ocular symptoms, the eye is not tender and the protrusion is reducible, we believe that the increase in the exophthalmos may be the result of the tenotomy of the fibrosed inferior rectus muscle.

COMMENT AND CONCLUSIONS

The most probable explanation of the mechanism by which the operation for pilonidal cyst with the patient under general anesthesia caused paralysis of the right superior rectus muscle, exophthalmos and complete fibrosis of the inferior rectus muscle was that an embolism of the artery supplying the superior rectus muscle occurred. Fibrosis of the inferior rectus muscle may have been caused by the secondary contracture associated with embolic myositis.

A specimen of the inferior rectus muscle was not obtained for biopsy because of extensive depression and fibrosis.

It was thought before operation that numerous adhesions existed between the muscle and the eyeball as a result of inflammation, but none was found.

There may be no proved causal relation between the operation with the patient under ethylene-ether anesthesia and the sudden onset of diplopia and the proptosis, but the short interval of less than twenty-four hours between the occurrence of the ocular symptoms and the operation is highly suggestive of a close relationship. It seems reasonable to believe that the condition of the eye could have been present some hours before it was noticed.

The severe vertical strabismus was remedied by complete tenotomy of the inferior rectus muscle, resection of 8 mm. of the superior rectus muscle, transplantation of the lower half of the lateral rectus muscle to the temporal half of the stump of the inferior rectus muscle and reattachment of the upper half of the lateral rectus muscle to the lower half of its stump. It was difficult to decide what procedure was indicated in this case, but the resulting useful field of binocular fixation indicates that the technic employed was reasonably sound physiologically.

35 East Seventieth Street.

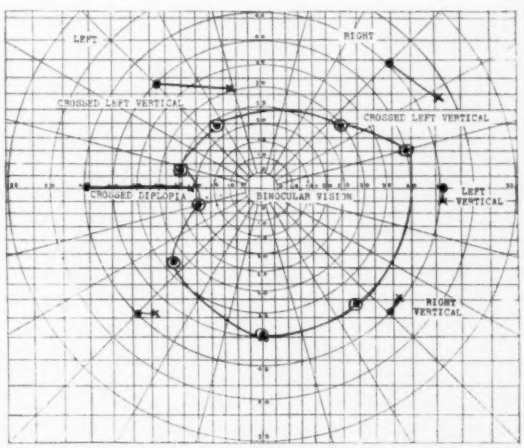

Fig. 4.—Field of diplopia and field of binocular fixation on March 1, 1943, three weeks following the operation. The amount and nature of the diplopia, plotted at 75 cm., was determined with a red glass over the left eye and the right eye fixing.

DISCUSSION

DR. CHARLES ALLEN PERERA, New York: Do the authors think there is any similarity between this case and the cases of orbital myositis reported before this section last year by Dr. Dunnington and Dr. Berke (Exophthalmos Due to Chronic Orbital Myositis, ARCH. OPHTH. **30:** 446 [Oct.] 1943)? The onset is different, but the clinical picture is the same and the observations at operation are similar. The suggestion of an inflammatory embolic cause in the case reported may give a clue to the cause of the orbital myositis.

DR. RUDOLF AEBLI, New York: What explanation is offered for the exophthalmos which is present, and why could the eye not be raised above the horizontal plane if there were no adhesions below at the time of the operation?

DR. RALPH I. LLOYD, Brooklyn: What anesthetic was used?

DR. ISADORE GIVNER, New York: How long after the appearance of the symptoms was surgical intervention carried out?

DR. GIOVANNI PACCIONE, New York: Was diabetes ruled out?

CAPTAIN GERALD FONDA, M.C., A.U.S.: To answer the last question first: Both the amount of sugar in the blood and the results of urine analysis proved that diabetes was not present.

Dr. Lloyd, anesthesia was induced with ether and ethylene.

Dr. Aebli, the eye cannot be elevated, even with fixation forceps, because of the extensive fibrosis of the muscle.

Dr. Perera, I believe that in chronic orbital myositis there is a progressive inflammatory process with fusiform enlargement of muscle, and there are at times adhesions between the orbit and the muscle. In this case no adhesions were discovered between the muscle and the orbit, and no fusiform enlargement of muscle was found. The myositis was acute and was localized in the inferior rectus muscle, resulting in fibrosis.

Dr. Givner, the operation on the eye was done thirteen months after the onset of ocular symptoms.

GLAUCOMA ASSOCIATED WITH NEVUS FLAMMEUS

REPORT OF A CASE

MAJOR ERWIN E. GROSSMANN

MEDICAL CORPS, ARMY OF THE UNITED STATES

The occurrence of glaucoma with nevus flammeus is uncommon, although not rare, in both the foreign and the American literature. The condition was first described by Schirmer in 1860, in association with buphthalmos, and since then several others have added to the picture to warrant its aeceptance as a clinical entity, manifested by nevus flammeus, glaucoma, epileptiform seizures, nevi and dilated vessels in the conjunctiva, iris and choroid.

Of the many observations made in cases of this syndrome, few have agreed concerning the mechanism of the glaucoma; indeed, etiologic theories have varied sufficiently to permit O'Brien and Porter [1] to state that the cause was unknown. From the evidence in many of the reported cases and in the present case, it appeared that perhaps another mechanism, not associated with increased intraocular pressure, may be the cause of the glaucomatous atrophy of the optic nerve.

REPORT OF CASE

The patient, aged 29, born in Wisconsin, was seen in the outpatient clinic on March 2, 1943. He presented no complaints but had come in for examination during a routine survey of all ocular defects at the Army Post. He gave the following history: He had had poor vision in the left eye since the age of 12 years, but medical attention was never requested. Since the age of 18 or 19 all vision, even perception of light, had been gone in this eye. He had had "fainting spells" since the age of 17 and had had three such attacks during the past six months. He was hospitalized twice, and a tentative diagnosis of epilepsy was made on his second admission to the hospital. He stated that he had never had any discomfort or pain in the left eye. The family history was noncontributory.

Examination revealed that the patient was physically sound, with a port wine nevus involving the left side of the face, including the forehead, temple and cheek. Both the upper and the lower eyelid were partially involved, as well as the lower palpebral conjunctiva and two thirds of the bulbar conjunctiva. One large tortuous conjunctival vessel coursed about the entire circumference of the cornea, about 1.5 mm. from it, giving off small branches to the limbal area. The cornea measured 12 mm. in the horizontal diameter, and the anterior chamber appeared to be of normal depth. The pupil was semidilated and reacted sluggishly to light. Slit lamp examination did not reveal any new vessels in the iris or any aberrations except for a remnant of persistent pupillary membrane, which extended from 9 to 12 o'clock. The lens and the vitreous were clear, and the fundus revealed a pronounced degree of glaucomatous cupping, the vessels bending at a right angle as they dipped over the scleral rim and soon being lost in a whitish, fluffy mass of what appeared to be scleral supporting tissue. There was no evidence of choroidal atrophy or signs of angioma. The vessels appeared to be normal in caliber and were less tortuous than normal. The tension was determined on two separate days and at different times of the day. Four readings showed 19, 21, 20 and 23 mm. (Schiøtz). Vision was nil, with no perception of light. The right eye was normal in all respects, and determinations of the tension made at the same time as those on the left eye showed 11, 14, 12 and 12 mm. (Schiøtz). The field of vision, both central and peripheral, was normal. Neurologic studies showed nothing abnormal and dermatologic examination revealed, in addition to the nevus described previously, a pea-sized hemangioma on the face near the hair line of the left temple.

Roentgenograms of the skull did not reveal any calcification of the meningeal vessels. Laboratory tests gave normal results. Both eyes were dilated with 2 per cent solution of homatropine hydrobromide, which was instilled three times at five minute intervals; the tension was determined before each instillation and, again, thirty minutes and one hour after the first instillation. No increase in tension was noted in either eye. The patient was given 1,000 cc. of water, and the tension was determined before and one hour after the water was drunk. No elevation of tension was noted in either eye, the difference between the first and the last reading varying only 1 mm. of mercury (Schiøtz). Gonioscopic facilities were not available.

COMMENT

No attempt has been made to review the literature, inasmuch as O'Brien and Porter [1] thoroughly covered the field in 1933 and Ehrlich [2] recently cited freely many of the other observers. The etiology was discussed fully by O'Brien and

From the Department of Ophthalmology, Regional Hospital, Camp Maxey, Texas.

1. O'Brien, C. S., and Porter, W. C.: Glaucoma and Naevus Flammeus, Arch. Ophth. **9**:715 (May) 1933.

2. Ehrlich, L. H.: Bilateral Glaucoma Associated with Unilateral Naevus Flammeus, Arch. Ophth. **25**: 1002 (June) 1941.

Porter. Ehrlich expressed the opinion that the occurrence of epileptiform seizures is exceptional, but the association is frequent enough to warrant one's anticipating this symptom in every case. Mehney [3] stated that usually nevus flammeus is not associated with glaucoma unless the nevus involves the lids or other ocular structures, and he speculated whether nevus involving these structures occurs without glaucoma.

In the case reported here, although deep glaucomatous cupping was present, no evidence of increased intraocular pressure was noted. Bär [4] described a similar case in which both eyes were involved; one showed partial excavation of the disk and the other merely a pale disk, but no glaucoma. Lowenstein [5] reported a case in which nevus flammeus occurred in a woman aged 40 who also had partial excavation of the disk but no elevation in tension; her visual fields and vision were normal. McRae [6] reported a case in which there was a goblet-shaped cavity in the optic nerve head but no increase in tension. He stated the belief that the condition was pseudoglaucoma. O'Brien and Porter reported a case of deep glaucomatous cupping with a tension of only 12 mm. (Schiøtz) and concluded that it was an instance of arrested hydrophthalmos.

Therefore the number of cases on record in which there was no evidence of glaucoma (increased intraocular pressure or instability of the pressure-regulating mechanism) but in which there was glaucomatous-like excavation of the disk is sufficient to warrant the assumption that the cupping is not always due to glaucoma but is simply, as McRae stated, pseudoglaucomatous. It does not seem logical to assume that glaucoma based on a congenital permanent defect, whether in the choroid or in the angle of the chamber, would in due time become arrested and finally manifest comparatively low tensions. The provocative tests performed in this case caused no embarrassment of the mechanism for the exchange of intraocular fluid. Yet the cupping of the disk in the left eye was of such a degree as is often seen only in the latest stages of glaucoma.

Of possible significance was the large amount of scleral supporting tissue seen within the abnormal cup. This suggests an anomaly within or about the optic nerve head, close to the cribriform fascia, which was weakened to such an extent that an intraocular tension as low as 19 to 23 mm. of mercury (Schiøtz) was sufficiently powerful to force it backward, dragging the nerve fibers along as they crossed the sharp rim of the scleral canal. The tension in the right eye was constantly from 7 to 13 mm. of mercury lower than that in the left eye, and this difference, together with the defect of the cribriform fascia, may have been sufficient to give rise finally to the typical glaucomatous cupping.

I do not offer the view that telangiectatic changes occurred behind the nerve head, for no abnormalities were grossly apparent, and the retinal and choroidal vascular systems seemed normal. It is more likely that the defect was congenital; several congenital anomalies frequently occur together, without necessarily being related embryologically—for instance, the persistent pupillary membrane in the left eye in the case reported in this paper was an associated congenital defect. It is entirely possible, therefore, that this syndrome is not always associated with glaucoma but that the latter is determined largely by the location of the congenital defect. I suspect that if gonioscopic studies had been made in all cases reported a defect would have been found in the angles of the chamber in the cases with associated buphthalmos and that such a defect would undoubtedly be related to a nevoid condition, directly or indirectly. In the cases in which the picture of adult glaucoma is presented, I should suspect the angles of the chamber to be open and functioning and the mechanism for the production of the glaucoma to be that for which Ehrlich expressed preference, namely that telangiectatic changes in the uvea, especially the choroid, result in disturbances in the exchange of intraorbital fluid. In those cases reported previously, including the present one, in which no increase in the intraocular pressure was observed, the condition may well be pseudoglaucoma. This assumption is strengthened by the negative results of the provocative tests performed in the present case.

CONCLUSIONS

The occurrence of a case of nevus flammeus with ocular changes resembling the late stages of glaucoma affords an opportunity for further speculation as to the mechanism of the glaucoma.

In all cases of this syndrome the type of glaucoma is not necessarily the same, but depends on the region which may be affected by a con-

3. Mehney, G. H.: Naevus Flammeus Associated with Glaucoma, Arch. Ophth. **17**:1018 (June) 1937.

4. Bär, C.: Ein bemerkenswerter Fall von Feuermal und Glaukom, Ztschr. f. Augenh. **57**:628, 1925.

5. Lowenstein, A., abstracted, Klin. Monatsbl. f. Augenh. **70**:540, 1923.

6. McRae, A.: Pseudoglaucoma, Brit. J. Ophth. **13**: 63, 1929.

genital anomaly; thus, buphthalmos results if the angle of the chamber is completely closed, and the adult type of glaucoma, if the angle is incompletely involved, together perhaps with other anomalies of the uvea.

A third mechanism may be that suggested in this case, in which no true glaucoma could be proved to exist; i. e., there was no typical increase in the intraocular pressure, but glaucomatous cupping resulted from a congenitally weakened lamina cribrosa, on which the exertion of a tension of 19 or 23 mm. of mercury produced as pathologic an effect as would a tension of 40 or 50 mm. of mercury in a normal eye.

Inasmuch as postmortem studies are infrequent in cases of this syndrome, gonioscopic examination would be invaluable in clarifying some of the conjectures concerning the mechanism involved in the production of glaucoma.

PATHOGENESIS OF GLAUCOMA

LEO HESS, M.D.
BOSTON

In two articles[1] a modest attempt has been made to give a more precise idea of the pathogenesis of the rise of intraocular pressure in acute glaucoma. This condition, according to my concept, does not arise primarily in the eyeball but has its origin in certain nerve structures outside the eye—the ciliary ganglion, the important diencephalic vegetative center (Karplus-Kreidl center[2]), at the base of the brain, in the vicinitiy of the optic chiasm and in the cortex of the brain. Mistakenly, the signs of acute and of certain kinds of chronic glaucoma were once considered to be inflammatory. Actually, the classic signs of common inflammation—arterial hyperemia, exudation and migration of white cells—cannot be demonstrated either by clinical or, to any significant degree, by histologic studies. The same holds true of the so-called inflammatory signs following thrombosis of the vena centralis retinae. If any inflammation exists in either instance, it is of a peculiar type ("angioneurotic inflammation"?). The sudden onset of the glaucomatous attack reminds one of an acute crisis of the vegetative nervous system, e. g., the epileptic fit or the rapid development of edema of the lungs in association with diseases of the brain or the spinal cord (Hess[3]). The actual site of the crisis in acute glaucoma is the ciliary ganglion and the nerves and capillaries of the ciliary body. It develops there under the indirect influence of the cortex (*glaucoma emotivo*, of the Italian school) or under the immediate influence of the diencephalic center, whence the irritation is conveyed to the bulbus by way of certain nerves, to be discussed later. The crisis consists primarily of a vasomotor and secondarily of a secretory disturbance (in passing it may be mentioned that in the epileptic fit, as well as in acute edema of the lungs, it is the vasomotor nerves that play an eminent role). The bulbus is the terminal organ. The crisis is a constrictor, or pressor, one. This can be deduced (1) from the narrowing of the terminal branches of the retinal artery after an attack[4]; (2) from the effect of miotics (Kadlicky, Thiel, Koller), resulting in vasodilation; (3) from the sudden rise of the systemic arterial pressure, which often antedates the attack; (4) from the arterial pulsation in the papilla after the attack, and (5) from the experimental evidence, provided by Ebbecke, that the impairment of eyesight during increased intraocular pressure is dependent on ischemia of the retina. The importance of the changes in the other tissues (cornea, lens, corpus vitreum, optic nerve) should, however, by no means be underestimated.

The mydriasis being unquestionably sympathogenic,[5] all the other signs of the acute attack, as has been pointed out in the second article,[1b] can be accounted for on a neurogenic basis: the headache, associated with nausea and vomiting, comparable to the headache and vomiting in cases of tumor cerebri and of migraine; the lacrimation, and the photophobia. The edema of the cornea (together with anesthesia, due to the edema) and the edema of the conjunctiva, lids and iris are likewise a nervous effect and are similar to angioneurotic edema (Quincke's edema fugax urticaria). The moderate rise of temperature, not infrequently met with in cases of acute glaucoma, can be traced to irritation of the heat center at the base of the brain, not far from the Karplus-Kreidl center (Ranson[6]). In addition to these symptoms, some indirect clinical signs and many pharmacologic observations could be referred to which are confirmatory, I believe, of the existence of a nervous factor underlying acute glaucoma. As an organ of sight, the eye-

1. Hess, L.: Pathology of Acute Glaucoma, Arch. Ophth. **26**:250 (Aug.) 1941; Pathogenesis of Acute Glaucoma, ibid. **32**:128 (Aug.) 1944.

2. Karplus, T. P., and Kreidl, A.: Arch. f. d. ges. Physiol. **129**:138, 1909; **135**:401, 1910; **143**:119, 1911; **171**:192, 1918.

3. Hess, L.: Wien. med. Wchnschr. **84**:285, 1934.

4. The connection between acute elevation of the systemic blood pressure and heightened intraocular pressure is evidenced by the clinical experience that immediately after delivery both pressures are high. After intravenous injection of epinephrine into the ear vein of the rabbit, both the intraocular pressure and the blood pressure are elevated. With hypertension of chronic renal disease the intraocular pressure is not high.

5. Mydriasis is not secondary to a primary rise of intraocular pressure (Heine, L.: Klin. Monatsbl. f. Augenh. **40**:25, 1902).

6. Ranson, S. W., in Harvey Lectures, Baltimore, Williams & Wilkins Company, 1936.

ball, the optic nerve and the primary and secondary optic centers within the brain represent one system. According to my concept, the eyeball, certain peripheral nerves, the ciliary ganglion and the diencephalic center also represent a unit concerned with the vital function of regulation of intraocular pressure.[7] This functional unit is finally governed, as are all the functions of all the visceral organs, by the cortex. The purpose of this paper is to elucidate this point of view and to show how it applies not only to acute glaucoma but to all other forms of increased intraocular pressure, acute or chronic, primary or secondary. It is an attempt at a general theory of the cause of rise in intraocular pressure, an attempt which has not been made in the literature. This theory of the neurogenic origin of intraocular pressure may better enable one to differentiate the types of glaucoma and to evaluate the means of treatment at one's disposal. High intraocular pressure, however, is not identical with glaucoma. The same can be stated of the high arterial pressure which is the outstanding clinical sign of "essential hypertension." The essence and origin of the latter are as unknown as are the nature and cause of glaucoma.

Obviously, one is confronted with a certain nervous mechanism regulating the production, inflow and outflow of the fluids of the eye. It may be compared to the mechanism which is thought to regulate the arterial pressure—a concept which has proved in recent years to be helpful in elucidating, differentiating and treating the various types of arterial hypertension.

In the production of the fluids of the eye two factors are to be borne in mind: 1. Mere transudation, largely dependent on the intracapillary pressure in the ciliary processes, an increase in which, according to Krogh, is associated increased permeability and increased escape of plasma. This hydrostatic pressure is opposed by the osmotic pressure of the protein bodies of the blood plasma.[8] 2. An apparently true epithelial secretion of the ciliary body, which is known to be influenced by physostigmine and atropine in the same sense as are true glands (Seidel). The latter fact would lead one to suppose that there exists an influence of the vegetative nervous system on the ciliary body. The transudation, as far as it is dependent on the intracapillary pressure, is likewise regulated by nerve impulses. The outflow of the fluids of the eye, mainly through the spaces of Fontana and the canal of Schlemm into the anterior ciliary veins, takes place finally in the superior ophthalmic vein, which is practically continuous with the cavernous sinus. Neither the superior nor the inferior ophthalmic vein is provided with valves, and it is generally impossible to state the direction of the blood flow within these veins. On the other hand, the sinus cavernosus is known to contain in its lateral wall the ophthalmic and the maxillary branches of the trigeminus. Hence it is likely that the blood stream within the ophthalmic and ciliary veins, and indirectly the inflow of the intraocular fluids, is connected with nerve impulses.[9] There is no evidence of a primary blockade to the outflow of blood in the onset of acute glaucoma. The primary disturbance is suggested in the increased production and inflow of fluids. The amount of blood within the bulbus is connected with the venous return to the right auricle. Any venous congestion—due to strain on the heart muscle, compression of the jugular vein, or even administration of miotics, with sudden hyperemia within the eye—may result in an attack of glaucoma. Finally, it should not be forgotten that the wall of the eyeball, through its elasticity, may exert a certain influence on the pressure in the eye.

FIRST (CILIARY) REFLEX ARC

Physiologic Aspect.—The regulatory nervous mechanism I have in mind is necessarily a reflex mechanism, consisting, as does any reflex, of three portions, an afferent (proprioceptive) limb, an efferent (motor or secretory) limb and a central (ganglionic) portion. The last may be located within the cerebrospinal axis (cerebrospinal reflex) or within the vegetative nervous system (axon reflex).

The afferent limb of this reflex arc is made up of fibers of the ophthalmic division of the trigeminus, which are distributed to all the tissues of the bulbus. Sensory stimuli, arising in any part of the eye, are conveyed by these fibers to ganglion cells of the ciliary ganglion.[10]

7. Normally the intraocular pressure is maintained at an almost constant level, with small oscillations corresponding to the rhythm of the heart beat and the respiration. This can best be explained by neuroregulation and not by hormonal influences.

8. Artificial increase in the osmotic pressure of the blood plasma is always followed by lowered intraocular pressure (Duke-Elder, W. S.: Brit. J. Ophth. **10**:513, 1926. Dieter, W.: Arch. f. Augenh. **96**:179, 1925).

9. As early as 1864 the great physiologist F. Goltz demonstrated a tone in the veins which was dominated by nerves.

10. The impulses travel thence to the gasserian ganglion, to the nuclei of the trigeminus and then to the thalamus (of the same and, mainly, of the opposite side). Finally, through interpolation of thalamic neurons, the impulses reach the cerebral cortex (somesthetic area in the upper part of the parietal lobe).

As was pointed out in the first article,[1a] neither within the uveal tract nor in the ciliary processes can ganglion cells be found. In my opinion, the ciliary ganglion, along with the numerous nerve fibers within the eye, is to be considered as one unit. The ciliary ganglion, embryologically a derivative of the gasserian ganglion (Keibel and Mall [11]), is a sensory organ, and the only one in the eyeball, since nowhere else can sensory terminations be detected within the bulbus (Stöhr [12]). This ganglion is the center for all the nerve impulses arising within the eye. It represents the central portion of the reflex arc under discussion.

It is essential to recall that the ciliary ganglion has the following connections: (1) With the parasympathetic nervous system, through its short root. By this path impulses are carried to the sphincter iridis and to the ciliary muscle. The radix brevis is an efferent, visceromotor bundle. (2) With the sympathetic nervous system, through its sympathetic root. These fibers derive immediately from the cavernous and the internal carotid plexus, but they originate in the ganglion cervicale supremum [13] and the centrum ciliospinale (Budge [14]) in the spinal cord (at the level of the sixth cervical to the third thoracic segment). These, too, are efferent fibers, innervating the dilator iridis, Müller's muscle and the vessels and capillaries of the eye.

The ciliary ganglion, being a part of the brain, shifted to the periphery by migration from the gasserian ganglion during embryonic life; the reflex arc is, therefore, a cerebral reflex.

Failure in the maintenance of the normal intraocular pressure may result from irritation of any one of the three components of this reflex arc.

Clinical Aspect.—Increased intraocular pressure may be due to the following factors:

I. Irritation of the Afferent Limb.

A. Diseases of the eyeball.

1. Cornea: (*a*) Keratitis punctata superficialis. In the second article [1b] I mentioned a case under my observation in which the condition started with small, painful erosions, followed by acute glaucoma. (*b*) Keratitis parenchymatosa, with episodes of high intraocular pressure. (*c*) Synechiae anteriores, particularly with complete adhesion of the pupillary part of the iris to the cornea.

It might be of interest to mention the "hypertonic reaction." After puncture of the anterior chamber in animals, the intraocular pressure starts to rise slowly and later increases rapidly up to 40 or 50 mm. of mercury; after oscillations the pressure returns to its normal level. Similar oscillations may be observed in patients.

2. Sclera: Scleritis profunda.

3. Iris: (*a*) Iritis serosa (glaucomatosa); (*b*) synechiae posteriores; (*c*) cyclitis.

4. Lens: (*a*) Cataracta traumatica; (*b*) cataracta senilis intumescens; (*c*) luxatio traumatica.

5. Retina: (*a*) Retinitis pigmentosa.

B. Involvement of the veins, with passive hyperemia of the eye.

1. Cardiac failure.

2. Engorgement of the veins due to local inflammation with occlusion of certain venous branches (tenonitis).

3. Thrombosis of venae vorticosae.

4. Periphlebitis in the vicinity of the eyeball.

5. Perforation of the internal carotid artery into the cavernous sinus.

6. Pressure of intraocular tumors on veins.

7. Thrombosis of the venae centralis retinae. Undoubtedly, the mechanical blockade to the venous flow is important. However, the cause of the secondary "inflammatory" changes with high intraocular pressure which may develop, without any signs of common inflammation, is entirely unknown. In the course of large intraocular hemorrhages, the intraocular pressure usually does not rise. Nor is the destruction of the retina, as a sequel of occlusion of the arteria centralis retinae, complicated with glaucoma. I may refer, with due caution, to this reflex. It may be of interest to recall that relief of pain and of congestion of the eye may sometimes be obtained by application of leeches to the temple; by withdrawal of blood and by use of cathartics. Noteworthy, as I said in the first article,[1a]

11. Keibel, F., and Mall, F. P.: Manual of Human Embryology, Philadelphia, J. B. Lippincott Company, 1912, vol. 2, p. 118.

12. Stöhr, P., Jr.: Mikroskopische Anatomie des vegetativen Nervensystems, Berlin, Julius Springer, 1928, p. 211.

13. Attention has not been paid in the ophthalmologic literature to the fact that the ganglion cervicale supremum is closely connected with an important blood pressure–controlling structure, the carotid sinus. A sudden rise of blood pressure, with its deleterious effect on the eye may on occasion be related to the carotid sinus. The role of the ganglion cervicale supremum in epinephrine mydriasis has been pointed out by me.[1c]

14. Budge, J.: Ueber die Bewegungen der Iris, Braunschweig, Fr. Vieweg u. Sohn, 1855.

is the intimate connection of the venous flow in the bulbus with many large venous plexuses in the vicinity (the reason for cauterization of the venous plexus at the middle turbinate, once advocated for treatment of glaucoma, can now be better understood).

8. Mydriasis, with pressure on the canal of Schlemm, and secondary venous engorgement. Severance of the afferent limb (iridectomy, Elliot's operation, cyclodialysis) will therefore lower the intraocular pressure.[15]

II. Irritation of the Efferent Limb.

1. Miotics, stimulating the parasympathetic portion of the reflex arc.
2. Ergotamine, paralyzing the sympathetic nerves, an effect which is equivalent to stimulation of the antagonist.
3. Epinephrine, acting as a stimulus to the sympathetic fibers. This effect is at first glance confusing. It proves, nevertheless, to be helpful in certain cases, not in those of the acute inflammatory type.[16]

III. Irritation of Central Part of the Reflex Arc.

1. Ciliary ganglion.

Of great interest from the point of view of this study is herpes zoster ophthalmicus. Aside from the well known involvement of the cornea, which may include all the tissues of the eye, there may be unilateral mydriasis, with lack of response to light. The tone of the bulbus is often lowered (Meller[17]), but in 1 interesting case (Urrets Zavalia[18]), increase in intraocular pressure, without any sign of obstruction of the angle, was the initial sign. The syndrome of unilateral mydriasis, with absence of reaction to light, and high intraocular pressure is striking and reminiscent of acute glaucoma. The primary site of herpes zoster ophthalmicus is the central portion of the sensory pathway of the eye, the gasserian ganglion. But in addition André-Thomas and Heuyer[19] demonstrated by microscopic studies severe active hyperemia and inflammation of the ciliary ganglion, i. e., of the central portion of the reflex arc. I feel justified in tracing the abnormal intraocular pressure associated with herpes zoster ophthalmicus to the involvement of the ganglion ciliare.

THE SECOND (DIENCEPHALIC) REFLEX ARC

By means of the regulatory mechanism outlined in the preceding section, all of the clinical signs of glaucoma in the affected eyeball can be accounted for. Here, one is confronted with an important question, for it is not one eye, but in most cases both eyes, which are involved sooner or later. With chronic glaucoma, even in the "normal" eye, the intraocular pressure may rise at night and the anterior chamber may be found to be shallow, the normal intraocular pressure being maintained, as has been mentioned, at constant level with but little oscillation, apparently owing to the activity of a regulating nerve center. When in cases of chronic glaucoma the function of this center is disturbed and the fluctuations of the intraocular pressure are pronounced, the possibility of a central nervous disturbance (anatomic or functional) must be considered. This concept seems to be confirmed by clinical and experimental facts.

Weekers[20] noted a rise in intraocular pressure in both eyes after cauterization of one eye. According to Thiel,[21] when the intraocular pressure falls in the injured eye, it drops for several days in the normal eye. Vom Hofe[22] observed bilateral lowering of the pressure after injection of a small amount of saline solution into one vitreous body.

Interesting are the studies by Kahler and von Sallmann[23] on patients with cerebral hemiplegia: On the paralyzed side the intraocular pressure was lowered.

15. Removal of the ganglion cervicale supremum (Abadie, C.: Bull. Soc. d'opht. de Paris, 1927, no. 9, p. 528. Lindsay, R. R.: Neuro-Ophthalmology, St. Louis, C. V. Mosby Company, 1941, p. 400) or roentgen irradiation (Thiel, R.: Klin. Monatsbl. f. Augenh. **82**:1909, 1929) would eliminate certain sympathetic influences. It could facilitate the action of epinephrine (Hess[16])—nothing more. It will soon be seen that a curative effect on glaucoma cannot be anticipated from this operation.

16. Hess, L.: Epinephrine Mydriasis, Arch. Ophth. **30**:194 (Aug.) 1943; footnote 1 *a*.

17. Meller, J.: Ztschr. f. Augenh. **43**:450, 1920.

18. Urrets Zavalía: Arch. de oftal. de Buenos Aires **1**:156, 1926.

19. André-Thomas and Heuyer: Rev. neurol. **20**: 381, 1912. André-Thomas: Le zona, Arch. Neurol. & Psychiat. **27**:216 (Jan.) 1932.

20. Weekers, L.: J. de neurol. **25**:778, 1925; Arch. Ophth. **41**:641, 1924.

21. Thiel, R., in Schieck, F., and Brückner, A.: Kurzes Handbuch der Ophthalmology, Berlin, Julius Springer, 1931, vol. 4.

22. vom Hofe, H.: Arch. f. Augenh. **102**:315, 1929.

23. Kahler, H., and von Sallmann, L.: Wien. klin. Wchnschr. **36**:883, 1923.

The occurrence of glaucoma after emotion has been noticed (Schoenberg [24]). Of importance are the pupillographic studies of Lowenstein and Schoenberg, demonstrating pupillary reactions of the "unaffected" eye in cases of unilateral glaucoma. Stimulation of the sympathetic trunk of one side gives rise to bilaterally increased intraocular pressure.

Papilian and Cruceanu [25] removed one hemisphere of the cerebellum in rabbits. On the side of operation the intraocular pressure remained unchanged or was slightly lowered; on the opposite side it was elevated.

It is in keeping with the physiology of today to search for a cerebral representation of any vegetative function. As was suggested in the second article, the subthalamic, diencephalic center of the vegetative nervous system (Karplus and Kreidl [2]) would provide the possibility of a bilateral nerve control of the eye. Stimulation of this center results in the acute experiment in protrusion of the bulbus, maximum mydriasis, widening of the palpebral fissure, lacrimation and pain—i. e., all the nervous signs associated with glaucoma. The pathways connecting this center with the nerves of the eye are the peduncles, the upper thoracic portion of the spinal cord and the centrum ciliospinale (Budge). Thence, the impulses spread to the ganglion cervicale supremum, to the cavernous and carotid plexuses and, finally, to the ciliary ganglion.

THIRD (CORTICAL) REFLEX ARC

Extensive clinical studies provided evidence (Hess and Faltitschek [26] that there are no vegetative centers in the cortex. Marburg [27] stated the belief that the subthalamic centers extend far upward to the medial aspect of the thalamus. The latter is connected with the cortex by the corticothalamic fibers. It is possible that the thalamus responds thus to cortical impulses (anxiety) and that through the thalamus the hypothalamic organ eventually becomes activated.

15 Kenwood Street, Brookline, Mass.

24. Schoenberg, M. J.: Role of States of Anxiety in Pathogenesis of Primary Glaucoma, Arch. Ophth. **23**:76 (Jan.) 1940.

25. Papilian, V., and Cruceanu, H.: Compt. rend. Soc. de biol. **92**:1081, 1925.

26. Hess, L., and Faltitschek, J.: Wien. klin. Wchnschr. **43**:488, 1930.

27. Marburg, O.: Verhandl. d. deutsch. Gesellsch. f. Nervenärzte **18**:208, 1929.

OPHTHALMOLOGIC FEATURES OF INTRACRANIAL CHORDOMA AND ALLIED TUMORS OF THE CLIVUS

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Intracranial chordoma and allied tumors of the clivus are of significance to the ophthalmologist, who may be the first to see the patient, since diplopia and visual disturbances occur in over a third of the cases and may, together with headache, be the only symptoms. In 1 case reported in the literature [1] muscle exercises were given before the true nature of the lesion was determined. The age incidence and the sequence of the symptoms may suggest that the tumor is chordoma, a diagnosis which can be further verified by roentgenographic and encephalographic studies.

That this suspicion may be of value to the neurosurgeon is easily understandable when it is realized (1) that tumors of this type are almost invariably fatal if left alone and (2) that surgical intervention in selected cases offers the only hope. If the surgeon suspects chordoma, he should not attempt complete removal but, rather, should employ suction of the gelatinous or mucinous content of the tumor, which, if successful will relieve pressure symptoms for a few years. If complete removal is attempted, death is likely to result.

A chordoma is a neoplasm arising from embryonic rests of the chorda dorsalis, a specific embryonal tissue about which the spinal column develops. As the base of the skull is molded, the posterior end of the cranial part of the chorda is forced backward and dorsally so that it lies on the occipital plate in a dorsal groove anterior to the foramen magnum. This embryonal tissue persists in infants in the centers of the intervertebral disks and in the coccyx, as well as at the base of the skull. Virchow [2] (1856) recorded the first description of a chordoma. In 1857, because of the physaliphorous nature of the tumor cells and because of his belief that they were of cartilaginous origin, he named the tumor ecchordosis physaliphora spheno-occipitalis. Müller [3] (1858) first stated that the tumor originated from embryonic rests of the chorda dorsalis. Ribbert,[4] in 1894, named the tumor chordoma. In 1897 Nebelthau [5] identified glycogen in the cytoplasmic vacuoles. The first case of symptomatic chordoma was reported by Klebs [6] in 1864. Grahl [7] studied the first spheno-occipital chordoma of clinical importance. Hass,[8] in 1934, published an excellent review of the literature on chordoma of the cranium and the cervical portion of the spine. The completeness of his bibliography makes it necessary to comment on reports made only after that date.

In this paper interest is directed only to spheno-occipital tumors and not to growths in the sacrococcygeal or vertebral area or in the region of the jaw. To date less than 100 cases of spheno-occipital chordoma have been reported. The benign form is of no clinical importance. The so-called malignant form is a slow, expansile growth, traversed by fibrous septums, between which is a sparsely cellular tissue composed largely of an intracellular matrix of gelatinous or mucinous character. The early structure resembles hyaline cartilage but is without intercellular substance. The cells are round, large, vacuolated and hyperchromatic and may resem-

Read at a meeting of the New York Academy of Medicine, Section of Ophthalmology, Nov. 20, 1944.

1. Carmichael, F. A.; Helwig, F. C., and Wheeler, J. H.: Cranial Chordoma: Report of a Case in Which Surgical Intervention Was Successful, Am. J. Surg. **55**:583 (March) 1942.

2. Virchow, R.: Ueber die Kretinen-Physiognomie, Verhandl. d. phys.-med. Gesellsch. **8**:24, 1857; Untersuchungen über die Entwicklung des Schädelgrundes, Berlin, G. Reimer, 1857, p. 51.

3. Müller, H.: Ueber das Vorkommen von Resten der Chorda dorsalis bei Menschen nach der Geburt und über ihr Verhältniss zu den Gallertgeschwülsten am Clivus, Ztschr. f. rat. Med. **2**:202, 1858.

4. Ribbert, H.: Ueber die Ecchondrosis physalifora spheno-occipitalis, Centralbl. f. allg. Path. u. path. Anat. **5**:457, 1894.

5. Nebelthau, L. A.: Ueber die Gallertgeschwülste am Clivus Blumenbachii, Inaug. Dissert., Marburg, 1897.

6. Klebs, E.: Ein Fall von Ecchondrosis spheno-occipitalis amylacea, Virchows Arch. f. path. Anat. **31**:396, 1864.

7. Grahl, O.: Eine Ecchondrosis physalifora spheno-occipitalis ungewöhnlichen Umfangs mit interessanten klinischen Folgen, Inaug. Dissert., Göttingen, W. F. Kaestner, 1903.

8. Hass, G. M.: Chordomas of the Cranium and Cervical Portion of the Spine: Review of the Literature with Report of a Case, Arch. Neurol. & Psychiat. **32**:300 (Aug.) 1934.

ble epithelium (fig. 1). The numerous variations in cell form, the occurrence of glandular alveolar structure, the morphology and staining reactions of the fibrils, the relation to cartilage and the taking of a red stain for glycogen are characteristic. The red stain obtained with carmine does not necessarily indicate glycogen, as the latter is soluble in water and is thus dissolved out in the solution of formaldehyde.[9] Ewing, however, stated that positive identification is not readily accomplished, as there is a close similarity between chordoma and myxochondroma. Because some of the cases in the literature of so-called chordoma are more than likely cases of myxochondroma, I have included the latter in this discussion, for clinically the two tumors are indistinguishable except for the more frequent occurrence of calcification in chondroma.

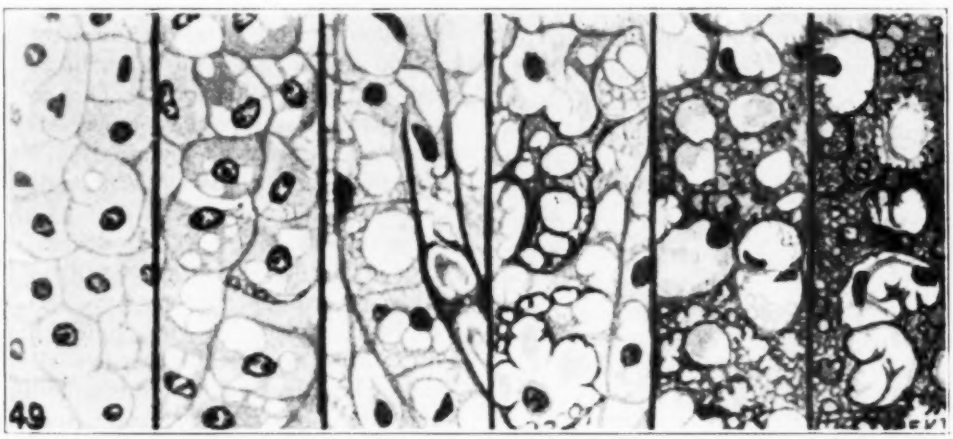

Fig. 1.—Transitions from the well preserved epithelioid tumor cells of chordoma through the physaliferous and signet-ring type of cell to extensive mucinoid degeneration (from Harvey, W. F., and Dawson, E. K.: Chordoma, Edinburgh M. J. **48**:713, 1941).

The majority of the tumors first cause symptoms in the third or fourth decade of life—the average age being 36 years. The first symptom can occur, however, at any age, from birth to 82 years. These tumors are found more frequently in males, the incidence being about 60 per cent. Characteristically they are located in the median line beneath the dura. Progression anteriorly is characterized by erosion of the sella turcica and compression of the optic chiasm. The osseous walls of the orbital cavities may be involved.

SYMPTOMS

The four most common symptoms of tumors of the clivus are headache, visual disturbances, nasal obstruction (from ventral extension of the tumor in the direction of the nasopharynx) and pain in the neck. The headaches are of increasing severity, with temporary periods of relief, and are referred to the frontal or the occipital region. Visual disturbances are the result of compression of the optic chiasm or of the sixth nerve.

Although the tumors are typically in the midline beneath the pons, there is a pronounced tendency for involvement of the cranial nerves to be unilateral, and even if the involvement is bilateral, it is more complete or widespread on one side than on the other. In 13 cases reported in the literature there were choked disks and in 10 optic nerve atrophy. Various defects in the visual fields simulated those caused by primary tumor of the hypophysis, but in the differential diagnosis there was lack of evidence of a functional disturbance of the hypophysis, and headache preceded changes in the visual fields; this is a reversal of the symptoms of tumor of the pituitary. Blurred vision, diplopia, internal strabismus, altered pupillary reaction, dilatation of the pupils, anisocoria and partial or complete ophthalmoplegia were a few of the various manifestations encountered on examination. Paresis of the abducens nerve occurred in 34 cases and was the most frequent symptom; it usually preceded the onset of paralysis of other ocular nerves. In the early stages paresis of the abducens nerve was predominantly on the left side, although in the late stages it was bilateral. Similarly, paralysis of the third and fourth nerves on the left side was more common than bilateral involvement. In almost every instance in which the facial nerve was disturbed the sixth nerve

9. Adson, A. W.; Kernohan, J. W., and Woltman, H. W.: Cranial and Cervical Chordomas: A Clinical and Histologic Study, Arch. Neurol. & Psychiat. **23**: 247 (Feb.) 1935.

was also involved. Practically any cranial nerve could be involved, with limited frequency.

Second in importance to involvement of the cranial nerves was the compression of the brain stem and the spinal cord in the region of the foramen magnum. Hemiparesis on the left side occurred in 6 of 7 cases. If the tumor was pushed through the foramen magnum, pain in the neck, which was accentuated by motion of the head, occurred. Van Wagenen [10] suggested that the frequency of symptoms and the onset are an aid in diagnosis. These symptoms in their order of frequency are as follows: palsies of the oculomotor nerves, with palsy of the sixth nerve as the most common; bilateral involvement of

Fig. 2.—Roentgenogram showing calcific flecks and increased density on the left side opposite the area of the sella turcica.

cranial nerves; symptoms of intracranial pressure, and involvement of the optic nerve.

Machulko-Horbatzewitsch and Rochlin [11] stated, in another way, that a typical course is characterized by involvement of the sixth cranial nerve followed by bulbar implication.

A roentgenogram indicates a destructive, nonproductive lesion of bone. Sosman, in a personal communication to Van Wagenen, pointed out a characteristic V-shaped notch in the basilar plate as an almost certain sign. A ventriculogram may show an elevated third ventricle.

The average duration of illness after involvement of the cranial nerves is about three years. However, cases in which the course was of eight

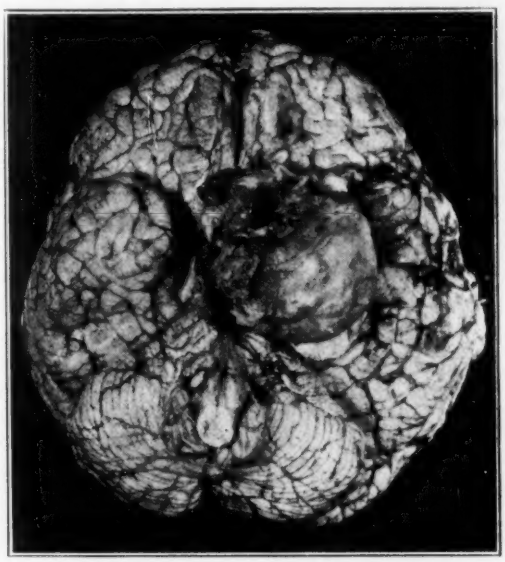

Fig. 3.—Under surface of the brain, showing the location of the myxochondroma.

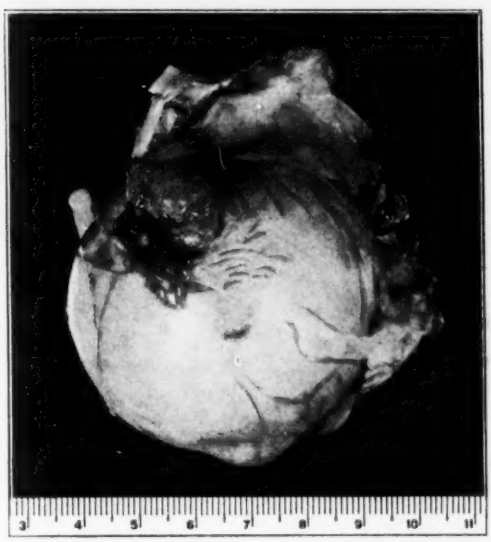

Fig. 4.—Tumor mass, 7 by 6.5 by 6 cm. The rule is in centimeters.

months to eighteen years' duration have been recorded.

In the differential diagnosis the following possibilities should be considered: tumor of the fourth ventricle, meningioma tumor of the cerebellopontile angle, infiltration tumor of the pons (glioma) and vascular lesions of the pons.

10. Van Wagenen, W. P.: Chordoblastoma of the Basilar Plate of the Skull and Ecchordosis Physaliphora Spheno-Occipitalis: Suggestions for Diagnosis and Surgical Treatment, Arch. Neurol. & Psychiat. **34:** 548 (Sept.) 1935.

11. Machulko-Horbatzewitsch, G. S., and Rochlin, L. L.: Klinik, Pathomorphologie und Histogenese der Chordome, Arch. f. Psychiat. **89:**222, 1930.

REPORT OF CASES

CASE 1.—R. W., a youth aged 20, was seen on March 18, 1941, through permission of Dr. Carl A. Peterson. His chief complaint was turning in of the left eye for the past ten months. The patient had previously been examined at St. Vincent's Hospital, five months after occurrence of this symptom. Complete examination, including roentgenographic study of the skull, revealed no abnormality at that time.

When the patient was first seen at the New York Post-Graduate Hospital, his vision was 20/20 in the right eye and 20/25 in the left eye; no change in the fundi or visual fields was noted. Paralysis of the left lateral rectus muscle was present.

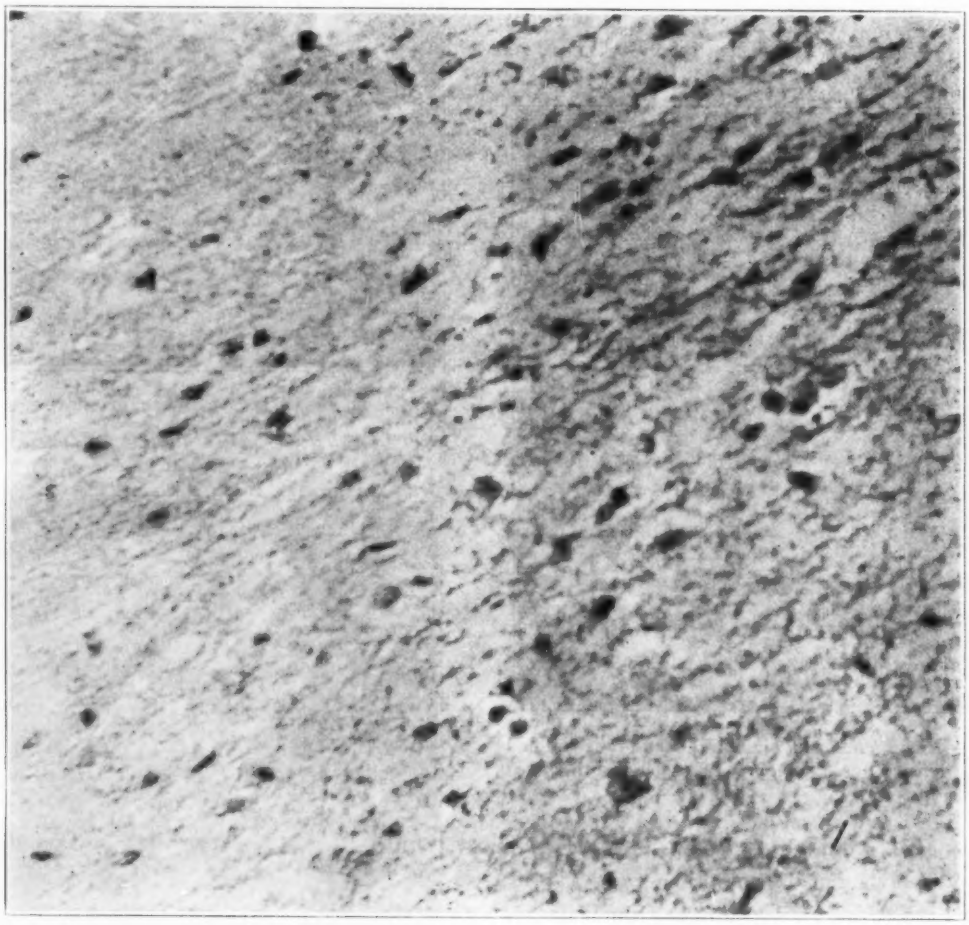

Fig. 5.—Photomicrograph of basophilic myxomatous tissue, with many moderately large, round, oval and stellate cells.

He was not seen again until Sept. 15, 1942. At that time he gave a history of difficulty in facial expression for four months. For the past month there had been weakness of his right arm. Headache had started about one year previously, and impairment of vision in his left eye had occurred about six months before. Since the onset of decreased vision in his left eye headache has ceased.

His right pupil was 5 mm. in diameter and reacted normally. There was loss of the temporal field of the right eye, suggesting that homonymous hemianopsia in the right eye had probably preceded the present state. His left pupil was 6 mm. in diameter and was fixed to light; no perception of light was present, and the fundus showed primary optic nerve atrophy.

General examination gave normal results except for paralysis of the left lateral rectus muscle and atrophy of the left optic nerve. The deep reflexes were greater on the right side, and the Babinski sign was present on that side. Sensory examination revealed nothing abnormal. A roentgenogram taken at this time showed an area of increased density, nearly 2 inches (5 cm.) in diameter, with numerous calcific flecks on the left side, opposite and extending somewhat posterior to the sella turcica (fig. 2).

An electroencephalogram indicated abnormal activity over both frontal lobes and the left occipital cortical area, with the abnormality more prominent over the latter region. A lesion in this area was suspected.

On Oct. 1, 1942 a decompression was done and the left middle fossa explored; a tumor was felt in the left temporal region, but it could not be exposed. The patient died the next day.

Diagnosis at necropsy was chondroma of the left spheno-occipital synostosis, with hemorrhage into the left lateral ventricle, the left cortex and the basal ganglions (postoperative). When the brain was removed, a tumor was uncovered, firmly attached to and apparently arising from the junction of the sphenoid and the occipital bone on the left side, between the

sella turcica and the foramen magnum. It measured 7 by 6.5 by 6 cm. (figs. 3 and 4) and arose 1 cm. to the left of the midline. From there it extended laterally into the left middle fossa, stretching and compressing the left optic nerve and compressing the overlying temporal and parietal lobes on the left side. The tumor was below the dura at the base of the skull. It was firm and, on section, was observed to be composed of grayish white, semiliquid, gelatinous tissue, with large areas of bone formation. Microscopically the tumor consisted of basophilic myxomatous tissue, in which there were many moderately large, round, oval and stellate cells (fig. 5). These cells had moderately dark, round and oval nuclei, with delicate chromatin network.

C. F., a woman aged 29, single, for the past two years had had diplopia and frequent headaches, particularly in the back of the head.

Examination on June 29, 1942 revealed that the pupils reacted to light, in accommodation and consensually. The left lateral rectus muscle was paralyzed. There was external ophthalmoplegia on the right side, due to involvement of the third nerve. Dr. Lowenstein made the following report: The deep reflexes on the right side were greater than those on the left; a questionable Babinski sign was elicited on the right side. The fundi and the visual fields were normal.

A ventriculogram made later revealed a concentric defect on the under surface of the third ventricle.

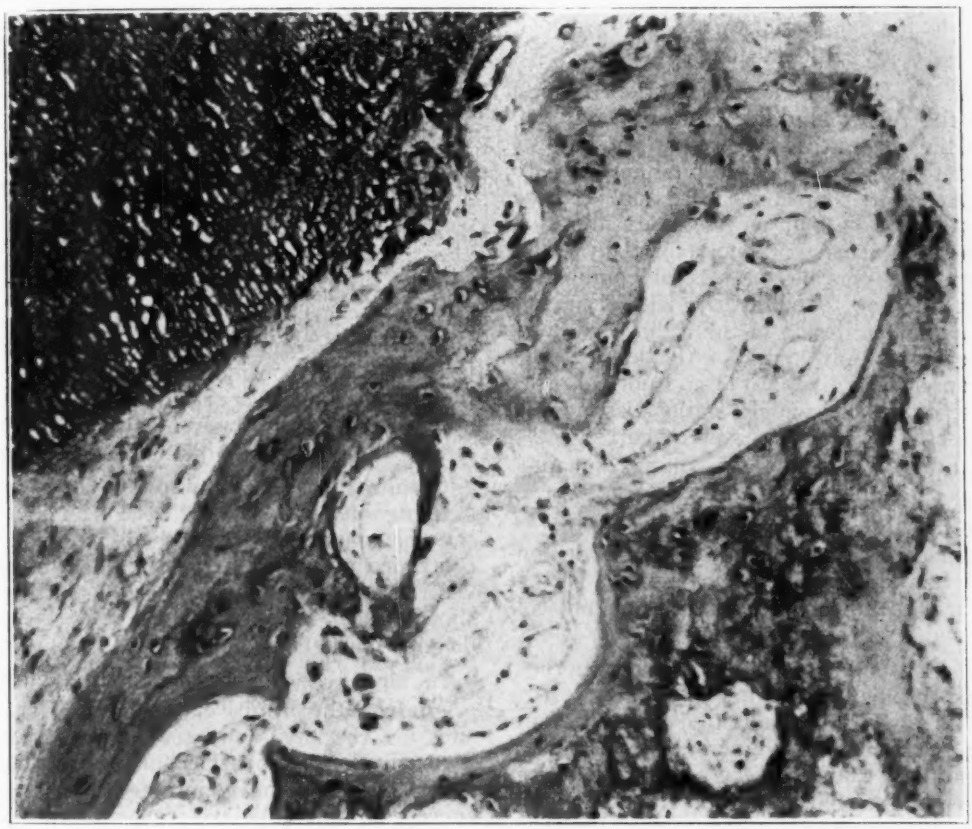

Fig. 6.—Photomicrograph of areas of bone and cartilage formation in the tumor.

Large areas of bone and cartilage formation were present (fig. 6). There was a limiting capsule of dense fibrous tissue. Histologically one cannot be certain whether this tumor was a chordoma or a chondroma. The pathologists consulted expressed a preference for the diagnosis of chondroma.

Case 2.—This case was observed through permission of Dr. Otto Lowenstein. The correct preoperative diagnosis was made by Dr. E. D. Friedman, who will report this case in detail at a later date. I refer to it here because the characteristic paralysis of the left lateral rectus muscle was an early sign and because postoperative histologic study verified the diagnosis of chordoma.

This corresponded with the position of the tumor mass, which was aspirated at operation, on Oct. 20, 1943. The histologic diagnosis was chordoma.

At the time of writing the right pupil was dilated (6 mm. in diameter) and did not react to light. Ptosis was present on the right side. The left pupil was 3 mm. in diameter and reacted to light. The patient held her head to the right because of the paralysis of the left lateral rectus muscle.

CONCLUSION

If the ophthalmologist suspects that an intracranial tumor is a chordoma, his impression

should be conveyed to the neurosurgeon, in order that the handling of the case may be materially altered.

Unexplained paralysis of the lateral rectus muscle, more frequently the muscle on the left side, in a patient in his thirties with progression to chiasmic signs, headache preceding defects of the visual fields and no evidence of disorder of the pituitary should suggest chordoma. Roentgenograms, encephalograms and ventriculograms, as well as subsequent bulbar involvement, may add corroborating evidence.

108 East Sixty-Sixth Street.

DISCUSSION

DR. E. D. FRIEDMAN, New York: I might amplify the report that Dr. Givner has already given of the second case. A woman aged 30 was admitted to the Bellevue Hospital on July 10, 1942. Her chief complaints were double vision, with strabismus of four or five years' duration, mild headache, urinary urgency (for many years) and occasional episodes of loss of equilibrium, with a tendenecy to fall to the left. She presented a complete lesion of the right third nerve (for the past year), palsy of the left external rectus muscle (for five years) and normal disks. The rest of the neurologic status was normal, and the serologic reactions were negative.

The results of roentgenologic studies, including encephalographic examination, were considered negative for tumor but were later reinterpreted, in view of the course of events. I shall demonstrate a few abnormalities which were present in the first roentgenograms but which my colleagues and I failed to evaluate properly.

The diagnosis on her discharge from the hospital was chronic encephalitis or syphilis. Nine months later she was readmitted because of increasing headache and dizziness, bouts of nausea and vomiting, weakness of the right arm and leg, numbness and tingling on the right side and difficulty in urination. Her general medical status was normal. The blood pressure readings were 130 systolic and 90 diastolic. No bruit was heard over the head.

Neurologic examination showed that visual acuity was somewhat impaired, but the fields were normal, and there was no papilledema. There was a lesion of the right third nerve, with a dilated, sluggish pupil. The left pupil was normal. There were weakness of the left external rectus muscle and slight weakness of the right side of the face. The tongue was deviated to the right. There was right hemiparesis, with increased tone and dragging of the right foot. Some cerebellar signs were present on the right side, with a tendency to fall to the right. The deep reflexes were more active on the right. All the abdominal reflexes were absent. Ankle clonus and the Babinski sign were elicited on the right side. Mild hypalgesia was present on the same side.

The urine was normal. Examination of the blood showed mild secondary anemia. The Wassermann reaction of the blood was negative. Blood chemistry was normal. The spinal fluid was under normal pressure; the total protein measured 100 mg. per hundred cubic centimeters. The Pandy reaction was positive and the Wassermann reaction negative.

We then reviewed the roentgenograms and encephalograms taken in 1942. In the lateral view of the skull, the dorsum sellae was eroded, and there seemed to be a calcified mass behind the sella in the region of the clivus. The first pneumoencephalogram showed a normal ventricular system, with no evidence of dilatation. In the encephalograms taken about nine months later a striking change was evident. The vestibular system had become dilated; the third ventricle, instead of being in the normal position, had been lifted up and was dilated and globular.

On the basis of the clinical history, the encephalographic findings and the simple roentgenograms of the skull, we ventured the diagnosis of chordoma. Aneurysm and meningioma at the base were also considered as possibilities, but I thought that aneurysm could be excluded by the absence of signs of involvement of the fifth nerve and by the presence of bilateral ophthalmoplegia.

Craniotomy was carried out by Dr. Joseph E. J. King on Oct. 20, 1943. Through a transcallosal approach he exposed a tumor which protruded upward through the floor of the third ventricle. Examination of portions of this tumor showed hard, calcified nodules. The pathologic diagnosis was chordoma.

As Dr. Givner has indicated, chordoma has its origin from the primitive notochord. The favorite sites are the clivus and the sacrum; the tumor is usually locally invasive but does not metastasize. There are no endocrine stigmas, such as one encounters in primary growths of the pituitary, and the symptoms indicate bilaterality. The ophthalmologic signs in this case appeared first on the left and later on the right; this is characteristic. There is usually a history of long illness but papilledema is not present. In this respect chordoma in this region behaves likes the so-called Schmincke tumor, which produces multiple cranial nerve palsies, owing to invasion of the base of the skull, but does not act like an intracranial tumor, which usually gives rise to papilledema.

Chordoma must be distinguished from aneurysm, the symptoms of which are seldom bilateral and are accompanied with signs referable to the fifth nerve, as has already been indicated.

I have now had the privilege of observing 3 cases of intracranial chordoma, the symptoms in all being somewhat similar. One makes the

diagnosis with hesitation, but it is occasionally a good guess.

DR. JOSEPH LAVAL, New York: Will Dr. Friedman say more about the differentiation of the early stages of chordoma and meningioma?

DR. E. D. FRIEDMAN, New York: I may simply say that we missed the diagnosis during our first observation of the case. We did not know what we were dealing with; we thought of syphilis but could not prove the diagnosis; we thought of chronic encephalitis, a diagnosis which is always dangerous and which the Germans call a "diagnosis of embarrassment." From the subsequent course of events, we felt that the case was one of a midline lesion exerting pressure on both sides. We were not inclined to the diagnosis of meningioma; meningioma usually produces unilateral signs. The long history, the bilaterality of signs and the roentgenographic evidence led us to favor the diagnosis of chordoma.

USE OF TRONCOSO'S MAGNESIUM IMPLANT IN CYCLODIALYSIS FOR RELIEF OF GLAUCOMA

OBSERVATIONS IN TWO CASES

P. H. BOSHOFF, M.D.
JOHANNESBURG, SOUTH AFRICA

The implantation of a strip of magnesium along the spatula tract in cyclodialysis is a modification suggested by Uribe Troncoso.[1] Because one is often forced to resort to cyclodialysis, one always feels that it is but a temporary measure. I wish to report on the postoperative course in 2 cases in which moderately severe reactions took place. In both cases the exact technic as described by Troncoso was followed except that a single, 6 by 1.5 mm., strip of magnesium was employed. This is less than the amount used by Troncoso.

Mr. F. R., aged 62 years, had a blind right eye due to thrombosis of the central retinal vein. The eye was glaucomatous, with an intraocular pressure of 54 mm. of mercury (Schiøtz). He refused enucleation, and it was decided to do an operation for implantation of magnesium in an attempt to retain the bulbus oculi. The strip was not inserted opposite 12 o'clock on the limbus because gonioscopic examination revealed engorgement of the vessels over the peripheral fold of the iris and a patch of enlarged capillaries on the surface of the adjacent ciliary body in the vertical meridian. In this congested patch capillary loops extended into the pectinate meshes of the uveal trabecula. The congested canal of Schlemm was visible as a pinkish line along the entire circumference of the angle. To avoid this area of engorged vessels, the magnesium was implanted opposite 10 o'clock, in the angle of the anterior chamber. The metal commenced to bubble immediately on insertion, and after twelve hours the whole anterior chamber was filled with gas. The cornea, however, remained clear in spite of a pressure of 50 mm. of mercury (Schiøtz). The iris was moist, and there was a fairly large subconjunctival bubble of gas. Because of the pain and because an obstruction to the escape of gas was thought to exist, a thin hypodermic needle was inserted through the cornea; the escape of the gas could be heard. Before deflation the eye was tympanitic when touched with the needle point. After deflation the anterior chamber immediately filled with a blood-stained fluid, and the pain subsided at once. This procedure had to be repeated daily for six days. After this there were a large bubble of gas in the anterior chamber and a similar bubble under the conjunctiva for three or four days.

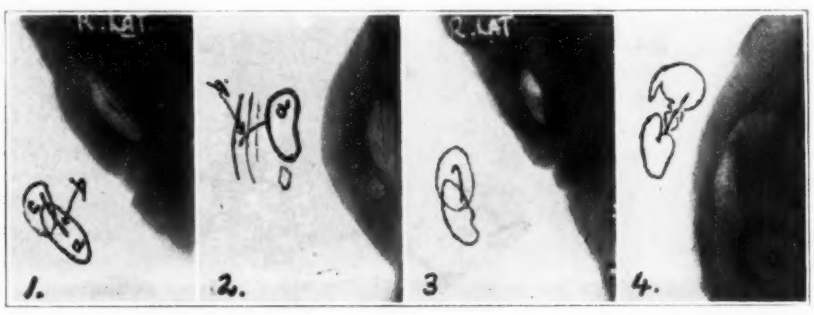

Fig. 1.—*1*, roentgenogram taken in Vogt's position, with the film pressed in between the nose and the eye. A soft tissue exposure is made from the lateral side. This shows (*a*) the anterior chamber of the eye filled with gas, (*b*) a thin dark line which is caused by the piece of magnesium, and (*c*) a subconjunctival bubble of gas.

2, roentgenogram taken in Clark's position, with the film pressed in between the lower edge of the orbital rim and the eye. A soft tissue exposure is made from above. After deflation there remains to be seen (*a*) a subconjunctival bubble of gas and (*b*) the shadow of the magnesium implant.

3 and *4*, roentgenograms taken in Vogt's and Clark's positions, respectively, two days after the roentgenograms *1* and *2*. The shadow of the magnesium implant is more indefinite (arrow).

Several interesting observations are worthy of record in this case.

1. Before the operation the cornea was hazy at an intraocular pressure of 54 mm. of mercury (Schiøtz), while the cornea was absolutely clear when the anterior chamber was full of gas, although the pressure varied from 49 to 55 mm. of mercury (Schiøtz). The haziness may be explained by the presence of corneal edema, due to aqueous being forced into the stroma through minute openings in the layer of Descemet.

2. On cocainization, prior to insertion of the hypodermic needle for escape of the gas, the cornea became insensitive within the normal period, but the iris remained intensely sensitive to being touched with the needle. This shows

1. Uribe Troncoso, M.: Cyclodialysis with Insertion of a Metal Implant in the Treatment of Glaucoma, Arch. Ophth. **23**:270 (Feb.) 1940; Tr. Sect. Ophth., A. M. A., 1939, p. 389.

that in routine cocainization the drug reaches the iris mainly via the circulation of the aqueous.

3. Tonometric readings showed that the intraocular pressure did not rise above 55 mm. of mercury (Schiøtz). I realize, therefore, that the magnesium stopped bubbling as soon as it was deprived of its surrounding fluid (aqueous). The result was that the pressure usually settled in the region of 50 to 55 mm. of mercury. On one occasion the patient was told to lie face downward, so that the aqueous could still surround the metal implant. Within two hours the intraocular pressure increased from 54 to 76 mm. of mercury (Schiøtz). This demonstrated that it was necessary for the patient to lie on his back so that the gas could collect in the anterior chamber and limit the increase of intraocular pressure by depriving the magnesium implant of its surrounding fluid.

The series of bone-free roentgenograms taken on dental film by the method suggested by Vogt [2] and Clark [3] clearly show how the entire anterior chamber is filled with gas, how the gas extends along the metal implant and how it collects under the conjunctiva. It can also be seen that the cyclodialysis tract is being kept open by the gas.

I was able to keep the patient under regular fortnightly observation for five months. The conjunctival congestion disappeared entirely during the three weeks which followed the operation, and the intraocular pressure remained between 24 and 29 mm. of mercury (Schiøtz) for almost five months, during which time the lens slowly became opaque and dilated vessels appeared on the iris. At the end of five months after the operation the patient returned, complaining of pain in the eye. The intraocular pressure was increased to 45 mm. of mercury. Gonioscopically, a complete circumferential iridocorneal synechia could be seen, and there was no suggestion of the presence of a surgical lesion in the angle. The amount of coarse pigment granules in the dependent part of the angle was in great excess. The eye was removed. Histologic examination of a meridional section of the enucleated eye showed that the tract of the filter had become closed and that a peripheral anterior synechia was present. One could not expect operation for hemorrhagic glaucoma of this type to be successful, but it is noteworthy that the ocular pressure had been kept normal for at least four and a half months by this improved method of cyclodialysis.

2. Vogt, A.: Skelettfreie Röntgenaufnahme des vorderen Bulbusabschnittes, Schweiz. med. Wchnschr. **53**: 982, 1923.

3. Clark, cited by Jackson, E.: Radiography of Foreign Bodies in the Eye, Am. J. Ophth. **5**:677, 1922.

A South African Negro, aged 38, had chronic noncongestive glaucoma in both eyes. Holt's iridencleisis was performed on the right eye, because gonioscopic evidence shows that in the Negro the trephine hole made in Elliot's operation usually becomes blocked with pigment granules. On the left eye Troncoso's operation for implantation of magnesium was performed, as previously outlined, after gonioscopic examination had shown that the iridocorneal angle was open in the vertical meridian. Although this case was not one of congestive glaucoma, the anterior chamber again became completely filled with gas, as in the preceding case, and a similar series of roentgenograms were obtained. The gas had to be allowed to escape by insertion of a hypodermic needle on four occasions. Tonometric readings were not taken, no tonometer being available at the clinic. The experiment of letting the patient lie with his face downward was again tried, and soon (in two hours) the patient complained of pain in the eye. Digital palpation confirmed the suspicion that the intraocular pressure was very high. After two weeks the eye was again normal in appearance. Examination after eight months showed no decrease in the peripheral visual field, and the intraocular pressure appeared to be normal on digital examination. Gonioscopically, the anterior opening of the cyclodialysis tract could be seen to have remained open. It is to be expected from experience with trephine operations

Fig. 2.—*1*, roentgenogram taken in Clark's position, showing (*a*) the subconjunctival bubble of gas, (*b*) the gas in the anterior chamber and (*c*) a well marked shadow of the magnesium implant.

2, roentgenogram taken in Clark's position two days later. This shows that the shadow of the magnesium implant (arrow) is more indefinite than that in roentgenogram *1*, owing to reduction in the amount of metal by the action of the aqueous.

that excessive deposition of pigment in the eye of the Negro may result soon in blocking the filter again.

I see no reason that Troncoso's method for implantation of magnesium should not be adopted in place of Heine's cyclodialysis. There are indications that by this means a more permanent filter is obtained. I am of the opinion that a single strip of magnesium measuring 5 to 6 by 1 mm. is the maximum amount of magnesium to be used, because of the tendency to excessive formation of gas. If this does occur, the gas can be allowed to escape simply by inserting a thin hollow needle through the cornea. Since this operation may be tried by other workers, I thought that it might be of value to record my experience in 2 cases.

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Clinical Notes

GONORRHEAL CHOROIDITIS TREATED WITH PENICILLIN

LIEUTENANT HERMAN KRIEGER GOLDBERG (MC), U.S.N.R.

Although there have been numerous reports of the efficacy of penicillin in treatment of external ocular conditions, none has indicated that inflammations of the posterior uveal tract are helped. Therefore it is well to report a case of choroiditis in which the result was particularly gratifying.

REPORT OF A CASE

In October 1943 a white man aged 30 was admitted to an overseas base hospital because of arthritis of the spine. While in the ward, he suddenly complained of blurring of vision in the right eye. After consultation with an ophthalmologist a diagnosis of acute choroiditis was made. Foreign protein in the form of triple typhoid vaccine was given, but there was no improvement. Because of the apparent chronicity of the inflammation, he was transferred to the Bainbridge Naval Hospital for further treatment. On his admission to this hospital the eyes were normal externally. Ophthalmoscopic examination of the right eye showed a hazy media, but no details could be seen. A vague outline of a large oval, yellowish area of exudative inflammation could be seen. The margins were indistinct, and the surrounding retina was edematous. This lesion was situated below and temporal but adjacent to the macula. It measured 2 disk diameters. Examination with the slit lamp demonstrated a moderate amount of pigment on the anterior capsule and an occasional precipitate on the posterior surface of the cornea, but there was no aqueous flare. Vision on his admission was 20/400 in the right eye and 20/30 in the left eye and was not improved with glasses.

The patient had been in excellent health except for an attack of gonorrheal urethritis seven years ago. The condition had been inadequately treated, but the discharge subsided spontaneously.

A complete examination of the patient failed to reveal any focus of infection. Examination of the teeth, nose and throat and general medical examinations revealed nothing abnormal. Agglutination tests for Brucella melitensis gave negative results. The white cell count was 10,000; the hemoglobin concentration was 96 per cent, and the urine was normal. The intradermal test with old tuberculin gave a negative reaction to 1 mg. Because of the history of gonorrhea and the presence of a strongly positive reaction to the complement fixation test for the disease and in the absence of any other focus of infection, it was thought that use of penicillin might be indicated. Therefore, the patient was given 160,000 units of the sodium salt of penicillin intramuscularly on each of three successive days. Improvement was noted after twenty-four hours. The media became clearer, and vision was improved to 20/70 at the end of twenty-four hours. Within ten days vision had increased to 20/40. The choroidal lesion lost all ophthalmoscopic evidence of activity. Some pigment was seen to be deposited at the margins of the lesion; the vitreous became completely free of opacities; the edema of the retina subsided, and the details of the fundus could be clearly seen. The choroidal lesion itself seemed to have resolved, and only a minimal amount of scar tissue remained. An additional 300,000 units of penicillin sodium was given two days later, and then its administration was discontinued. Vision has remained 20/40, and there has been no tendency of the inflammation to recur. The maximum improvement occurred within the first ten days.

SUMMARY

Penicillin had been used in 5 other cases of choroiditis, in which the cause was probably tuberculous. There was no improvement, but in this 1 case, in which the etiologic agent seemed to be the gonococcus, the result was spectacular. This response can well be correlated with the specific reaction of other forms of gonococcic infection to penicillin. Penicillin should not be used in the treatment of choroiditis unless the lesion is presumed to be due to gonorrhea.

From Bainbridge Naval Hospital, Bainbridge, Md.

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Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Blind

THE ARMY REHABILITATION PROGRAM FOR THE BLINDED AND THE DEAFENED. C. C. HILLMAN, J. A. M. A. **125:** 321 (June 3) 1944.

Hillman recounts the procedure employed for the rehabilitation of the blind. The public idea as to the use of guide dogs is that every blinded person should have one. Experience shows, however, that only about 10 per cent of the blind can use dogs advantageously. Many blinded soldiers will develop the ability to get about with the minimum of assistance. With a skilfully used cane, they are inconspicuous and unencumbered. Some persons do not like dogs; others are temperamentally unfitted for canine companionship. It is important to have determined the type of work to be done and whether a guide dog will be practical and compatible with the future choice of a job. Even when the use of a dog is contemplated, it is better for the blinded person first to develop his complementary senses to the fullest extent. The Veterans Administration and the Medical Department of the Army concur in the belief that a veteran should secure a guide dog only when it is shown that the dog is the best solution of the veteran's problem. Private guide dog agencies have agreed to provide dogs to blinded soldiers, either gratis or at nominal cost. It appears that the facilities of these agencies will be adequate to meet all anticipated needs and that governmental aid in supplying dogs will not be required.

W. ZENTMAYER.

Color Sense

PERCEPTION OF COLOR SIGNALS BY PERSONS WITH COLOR ANOMALIES UNDER PRACTICING CONDITIONS. N. WISHNEVSKY and A. FLEKKEL, Sovet. vestnik oftal. **22:** 16, 1943.

Since from 4 to 10 per cent of young men suffer from congenital disturbance of color sense, the authors consider the possibility of using these persons in aviation. They made tests with color signals in order to find whether candidates with anomalies for color can distinguish colored lights in aviation service, particularly in night flights. The subject was tested with three types of signals: (*a*) colored rockets (red, yellow, green, white) used in aviation, (*b*) aeronavigation lights and (*c*) colored signals. A special color signal apparatus was used, and the colored lights were observed on the ground and from the sky, at a height of from 500 to 1,000 meters. The color sense of the subject was examined with colored plates (Stilling, Ishihara, Robkin) and with Nagel's anomaloscope. The subject registered his findings himself during the night flight, and the records were controlled by the attached physician, whose color sense was normal. Forty-one young subjects were examined. Of these, 29 had deuteranomalopsia; 7, extreme deuteranomalopsia; 1, deuteranopsia; 1, protanomalopsia, and 3, protanopsia. Twenty-one were pilots: 12, co-pilots, and 8, radio gunners. The majority had from 50 to 500 flying hours of experience. The authors summarize their study as follows:

Classification of disturbances in color sense according to the Kries-Nagel scheme can be made on the basis of pigment tests and of examination with Nagel's anomaloscope.

Comparison of the data obtained with the pigment tests, the anomaloscope and the color signals and color rockets showed that the majority of mistakes were made by subjects with color blindness and to a lesser degree by the subjects with color anomalopsia.

Tests with the yellow and green rockets showed that even the control subjects made mistakes.

The colored signals were incorrectly identified from the ground and from the air by the subjects with color anopsia, by those with extreme deuteranomalopsia and by those with other forms of anomalopsia, in that order.

The identification of the colored signals during flying is much more difficult than in railroad and automobile transportation because of the small visual angle from which the signal is seen and the short time permitted for distinguishing the signal.

The application of colored signalization in aviation requires the correct differentiation and naming of the color.

O. SITCHEVSKA.

Congenital Anomalies

THE MARCUS GUNN PHENOMENON: REPORT OF A CASE. V. RAMÍREZ ESTEVA, Arch. Asoc. para evit. ceguera México **1:** 163, 1942.

The author considers this condition as exceedingly rare, since only about 100 cases have been reported since Marcus Gunn presented his first case before the Ophthalmological Society of the United Kingdom, in 1883.

The author describes the clinical aspects and possible factors in the causation, diagnosis and treatment of the disturbance and reports a case

of the syndrome in a man aged 23. The condition was congenital. Partial ptosis of the upper lid was present, and when the patient opened his mouth there was an involuntary elevation of the right upper lid. A moderate spasm of the right inferior rectus muscle was also present.

W. ZENTMAYER.

Cornea and Sclera

BLUE SCLERAS, BRITTLE BONES AND DEAFNESS: REPORT OF AFFLICTED FAMILY. J. E. FARBER and A. E. MARGULIS, Arch. Int. Med. **71:** 658 (May) 1943.

Farber and Margulis observed a family of 52 members, 12 of whom presented the syndrome of blue scleras. Seven of these patients have brittle bones; 4 are deaf, and 1 has only blue scleras. Eight are males and 4 females. The condition is transmitted and occurs equally in the two sexes. The physical attributes of small stature, hypermobility of the joints, relaxation of the ligaments and abnormal shape of the head were observed in the afflicted members. Three members of the group were studied in detail. Roentgenograms of the skeleton revealed slender bones, generalized osteoporosis and deformities resulting from multiple fractures.

J. A. M. A. (W. ZENTMAYER).

CLEARING OF VARIOUS CORNEAL OPACITIES: PRELIMINARY REPORT. E. G. LAZAREV, Vestnik oftal. **22:** 7, 1943.

The metabolism of the cornea, the cause of leukoma and the role of corneal transplantation in clearing adjacent corneal opacities are discussed in detail. Lazarev made clinical experiments in order to determine the role of excision of the cornea on the clearing of the opacities.

Seven cases of central leukoma of various origins are reported. In 4 cases (5 eyes) a strip of opaque cornea, 2 mm. wide and 4 mm. long, was excised. In 3 cases the excision of the strip was incomplete; i. e., it remained attached at one end and hung like an apron. In 5 cases vision improved from ability to see hand movements to 0.1; the blood vessels thinned and in some cases they disappeared. In 1 case of recent interstitial keratitis the operation was unsuccessful.

Lazarev thus considers excision of the superficial layers of the opaque cornea (1) as the first stage of penetrating transplantation of the cornea and (2) as a favorable factor in clearing corneal opacities.

O. SITCHEVSKA.

TREATMENT OF SERPIGINOUS AND OTHER PURULENT ULCERS OF THE CORNEA WITH SODIUM SULFANILAMIDE. A. GOLDBERG, Vestnik oftal. **22:** 34, 1943.

The author used local applications of the soluble sodium salt of sulfanilamide in treatment of 90 patients with keratitis and hypopyon and various other purulent ulcers of the cornea. Sulfanilamide powder was used every three hours the first twenty-four hours and then every three hours during the day until complete epithelization had occurred (no staining of the cornea with fluorescein). Instillations of a 30 per cent aqueous solution of the drug were then used four to five times a day until all inflammatory symptoms disappeared.

In 81 per cent of patients the ulcer was located in the center of the cornea, and in the majority it was deep and large (from 2.5 to 6 mm.). Bacteriologic examinations showed pneumococci in 86 per cent of the eyes. The majority of the patients entered the hospital from six to thirty days after the onset of the disease; nevertheless, the clearing of the ulcers was noticeable on the second or third day after initial treatment. In 66 per cent of patients complete epithelization of the ulcer was observed in from five to ten days. The hypopyon disappeared in many patients in from one to four days. The final visual acuity of 62 patients was rather high; in 50 patients it was increased from 0.06 to from 0.1 to 0.7.

The results thus obtained in treating purulent corneal ulcers with sodium sulfanilamide are encouraging. The method is a simple and radical one, with no complications. The purulent lesion begins to regress almost immediately; the epithelization of the ulcer is rapid, and it may be assumed that sulfanilamide activates the regenerative process of the cornea. O. SITCHEVSKA.

Experimental Pathology

CONGENITAL MALFORMATIONS OF THE EYES INDUCED IN RATS BY VITAMIN A DEFICIENCY. J. WARKANY and E. SCHRAFFENBERGER, Proc. Soc. Exper. Biol. & Med. **57:** 49 (Oct.) 1944.

The authors observed congenital defects of the eyes in the offspring of rats raised on a diet of ground whole wheat, crude casein, brewers' yeast and sodium chloride (diet U) supplemented with very small amounts of carotene and bred on a diet entirely free of carotene and vitamin A (diet W). Diet U made possible slow growth and maturation but no storage of the vitamin. If the growth curves of the rats became flat during this preparatory period, which happened three or four times, each rat was given 5 Gm. of horse muscle.

Twenty female rats of a certain strain were raised on diet U until they reached a weight of 150 to 160 Gm. and regular estrus cycles were established. Then they were placed on diet W, entirely void of vitamin A, and bred to males which had been fed an adequate diet. Within a week the females fed diet W showed pronounced signs of nutritional deficiency, and only 3 mothers carried their litters to or near term,

while 9 females resorbed their embryos. The 3 litters, obtained by cesarean section, consisted of 19 dead young, all of which had abnormal eyes. In 14 of these young the abnormality could be recognized by external examination. Instead of the closed eyes seen in normal newborn rats, "open eyes" of a reddish color were seen. Histologic examination showed that the eyes of all 19 young were abnormal. There was no clear differentiation between lids and cornea, and the anterior chamber was present in a rudimentary form only. The vitreous chamber was filled with connective tissue. The retina was folded and disorganized. In several specimens a coloboma of the retina was present.

As controls, 62 females of the same strain were fed diet W supplemented with vitamin A. They had 410 young, none of which showed abnormal open eyes.

P. C. KRONFELD.

General Diseases

TREATMENT OF LEPROUS SCLEROKERATITIS. T. APARISI, Arch. Soc. de oftal. hispano-am. **3:** 461 (Nov.-Dec.) 1943.

The most frequent ocular lesion of leprosy is sclerokeratitis. The condition is serious, and, in spite of all treatment, it sooner or later ends in destruction of the globe. Besides the routine treatment, Aparisi uses calcium in large doses, as though the lesions were of tuberculous origin. Although calcium does not cure the sclerocorneal process, it checks the progress to some extent and delays the destruction of the anterior segment.

In the routine medical treatment, protein therapy, autohemotherapy, chemotherapy of gold salts and arsenicals, fever therapy (intravenous typhoid vaccine), administration of sodium salicylate by mouth and local application of atropine, ethylmorphine hydrochloride and chaulmoogra oil are employed.

In the incipient form of the disease surgical procedures are used to some advantage. Curettage of the affected zone and coverage with a conjunctival flap have been of benefit.

H. F. CARRASQUILLO.

OCULOGLANDULAR TULAREMIA. N. A. PLETNEVA, Vestnik oftal. **22:** 11, 1943.

The literature, the clinical picture, the diagnosis and the differential diagnosis are reviewed in detail. Professor Pletneva stresses the identity of Parinaud's conjunctivitis and oculoglandular tularemia. The agglutination test is the most important diagnostic means; it gives a positive result in the second week, and the blood contains agglutinins for many months, and even years. The cutaneous allergic test with tularin is the next most important clinical diagnostic method. The disease is transmitted not only by skins of rabbits, rats or squirrels, but by dogs, horses and and cows and by bites of fleas, mosquitoes and horseflies.

The cases of 4 patients are reported. All were acutely ill, with chills and fever, and the preauricular gland was swollen in all. One patient had a few firm, semitransparent nodules on the conjunctiva, resembling a tuberculous lesion. Biopsy showed only a chronic inflammatory process. For 2 patients the diagnosis was difficult to make, as the lesion (a papule) was in the region of the lacrimal sac and a diagnosis of dacryocystitis was made; 1 of the patients was scheduled for extirpation of the sac, and only when the preauricular, submaxillary and cervical glands became inflamed and an agglutination test was done was the correct diagnosis arrived at.

Therapy is symptomatic; mercury ointment for the ulcers of the skin and convalescent serum are considered of great value in treatment of the oculoglandular form.

O. SITCHEVSKA.

Glaucoma

ANGIODIATHERMY OF THE LONG POSTERIOR CILIARY ARTERIES AND ITS USE IN THE TREATMENT OF GLAUCOMA. D. GUERRY, Am. J. Ophth. **27:** 1376 (Dec.) 1944.

Guerry gives the following summary:

"1. The results of coagulating the long posterior ciliary arteries of rabbits, singly and together, are compared with those obtained by Wagenmann after cutting these vessels.

"2. Coagulation of a single long posterior ciliary artery was found to reduce the intraocular pressure in both the rabbit and human for a period of about two weeks. The procedure is relatively harmless.

"3. Coagulation of both long posterior ciliary arteries in the rabbit resulted in phthisis bulbi in 50 per cent of the cases.

"4. The procedure is suggested as a rational substitute for posterior sclerotomy, and as a method of preventing the initial tension rise seen so frequently after cyclodiathermy.

"5. The technique of coagulating by diathermy the nasal long posterior ciliary artery in the human is described."

W. S. REESE.

GLAUCOMA AFTER CATARACT OPERATION. B. C. SHARMA, Indian J. Ophth. **4:** 51 (July) 1943.

Sharma states that in a series of 400 cataract extractions in which a conjunctival flap was made not a single instance of glaucoma occurred, whereas in 200 cases in which the incision was made in the cornea without a conjunctival flap 14 instances of glaucoma were noted. He concludes that whenever possible it is advisable to dissect a conjunctival flap in operations for cataract extraction. If glaucoma occurs within a few days after the operation, a paracentesis is done; if this fails, an Elliot trephine operation or a cyclodialysis is performed. The result is often

a failure, especially if the glaucoma is due to the growth of corneal epithelium into the anterior chamber.

W. ZENTMAYER.

Injuries

INTRAOCULAR FOREIGN BODY. R. OLIVERA LOPEZ, Arch. Asoc. para evit. ceguera México **1:** 143, 1942.

Olivera Lopez reports a case in which a fragment of stone or steel passed entirely through the cornea, producing a wound with herniation of the iris and diffuse opacities of the lens. A roentgenogram showed the foreign body at the apex of the orbit. One and one-half months later the lens was clear, and examination revealed a black plaque near the posterior pole of the fundus. A panoramic photograph of the fundus established the position of the foreign body, and the fragment was removed by electromagnet through a posterior sclerotomy. The resulting visual acuity was 5/10.

W. ZENTMAYER.

GUNSHOT WOUNDS OF THE ORBIT. A. B. KATZNELSON, Vestnik oftal. **22:** 13, 1943.

At a large hospital at the front orbital injuries constituted about 14 per cent of all injuries of the eye. This proportion, however, increased to about 50 per cent in the hospitals at the rear as a result of the improved diagnostic means, particularly the roentgenoscopic examinations. Since mines and grenades are widely used in this war, injuries of the face and eyes with fragments are much more common than those due to bullets, the ratio being about 77 : 23. Of the neighboring parts of the face, the nose, and especially the sinuses, were injured in 56 per cent of cases. In 25 per cent the fragments were found on the opposite side of the face. Frequently a fragment breaks into a few smaller ones and may injure distant structures.

Isolated injury of the orbital wall was seen in 66 per cent of cases, combined injuries in 34 per cent and injury of both orbits in 11 per cent.

Katznelson bases his classification of gunshot wounds of the orbit on (1) the course and length of the injury canal and (2) the relation of the foreign body to the orbit. The axial penetrating injuries usually cause crushing of the eyeball and its loss. The incidences of injuries of the eyeball in orbital wounds were about as follows: penetrating injuries to the eye with loss of the eyeball, 47 per cent; contusion changes, 35 per cent; neuritis, with evulsion of the optic nerve, 6 per cent; ruptures of the choroid and retina, 26 per cent, and no changes in the eye, 9 per cent. Ruptures of the choroid and retina were often the result of contusion injuries. The retinal tears might be multiple. Retinal detachments were rarely observed with simultaneous multiple ruptures of the choroid. The chief observations of Lagrange on contusion changes in the eye were confirmed in this war, except that choroidal ruptures were observed with tangential injuries of the orbit and that with injuries of the upper orbital wall there were also contusion changes in the eye. There were seen evulsions of the optic nerve, neuritis, optic nerve atrophy and optic neuritis with central scotoma due to injuries of the sphenoid and ethmoid sinuses. A peculiar syndrome—slight exophthalmos, ptosis, complete ophthalmoplegia and trophic degeneration of the cornea—was observed in cases of fracture of the upper orbital fissure, hemorrhage or direct injury of the nerves passing through this fissure.

Treatment concerns only the first aid for and early therapy of gunshot wounds of the orbit. Since gas gangrene develops rarely in the face, primary sutures are indicated in injuries of the lids and conjunctiva. It is essential to preserve all the lid and conjunctival tissue, as it will avert the necessity of plastic operations in the future. All small fragments of bone which have the periosteum and soft tissues partly intact should be preserved, as they will serve for the reconstruction of the orbit later. In cases of severe injuries, with fracture of the walls of the ethmoid and maxillary sinuses, the wound is left open for drainage; accurate débridement should be done. If fragments penetrate the sinuses and there is danger of infection of the meninges, sulfanilamide powder should be used locally and the drug administered internally. In retrobulbar hemorrhages with exophthalmos it is wise to suture the lids in order to prevent ulcerative keratitis. In severe injuries of the orbit it is advisable to do an early enucleation. In large defects of conjunctival tissue no sutures should be applied, as the indication for early prosthesis is much greater. If a discharge is present after enucleation, a fistula connecting with one of the sinuses or a foreign body should be looked for.

The article is an extensive and thorough analysis of gunshot wounds of the orbit. A number of case reports give a wealth of information concerning the extensive and varied injuries of the orbit observed in the present war.

O. SITCHEVSKA.

Lens

JUVENILE CATARACT IN ASSOCIATION WITH DERMATOSIS. D. M. ROLETT, Am. J. Ophth. **27:** 389 (April) 1944.

Rolett reaches the following conclusions with regard to the disease described by Rothmund:

"The important points to be remembered are:

"1. Occurrence of cataractous changes in rather young people with typical skin changes which may or may not arise simultaneously.

"2. Evidence of pluriglandular endocrine disturbances.

"3. Skin changes although bearing a similarity are not always of the same origin and are not therefore the cause of the disease.

"4. The development of the cataract is rapid and progressive.

"5. The prognosis as to the operation and subsequent vision is favorable.

"6. Fortunately the disease is not common.

W. S. REESE.

A MODIFICATION OF THE CORNEAL SECTION IN THE OPERATION FOR CATARACT. J. H. BAILEY, Am. J. Ophth. **27:** 1253 (Nov.) 1944.

Bailey gives the following summary:

"An operation for cataract is described in which the corneal flap is fashioned by an incision from without inward and perpendicular to the plane of the globe. The advantages of the procedure are enumerated and explained. The instrument employed is a short Bard-Parker handle and an attached blade which is keen, inexpensive, and replaceable." W. S. REESE.

Methods of Examination

A SIMPLE METHOD OF DEMONSTRATING NYSTAGMUS IN CERTAIN MINERS. A. C. REID, Brit. J. Ophth. **28:** 598 (Dec.) 1944.

This note is designed to fill a gap in the diagnosis of a form of nystagmus which it is difficult to demonstrate when the man sits upright in a chair but which results in definite distress at work involving stooping. The miner states that as soon as he straightens up the oscillations cease. Even a rapid approach with the ophthalmoscope cannot catch them. To get around this, the author employs a concave mirror 12 cm. in diameter with a curvature of — 0.75 D.

The patient stoops while sitting or kneeling and fixes his gaze on the mirror; when he exclaims that the movements have begun, a beam from a pocket torch is directed on the eye being examined, and the oscillations, magnified, are seen at once in the mirror by the observer.

W. ZENTMAYER.

Neurology

MYASTHENIA GRAVIS OF OPHTHALMOPLEGIC TYPE. J. M. ZAVALIA, M. B. ZURBRIGGER and O. RUSSO, An. argent. de oftal. **4:** 144 (Oct.-Dec.) 1943.

Two cases are reported to stress that myasthenia gravis should be considered in cases of ophthalmoplegia and that there is a simple pharmacologic test to confirm the diagnosis.

The cases presented are those of 2 children, 3 and 8 years old. Both patients presented bilateral external ophthalmoplegia, which was less conspicuous in the morning and became accentuated in the evening. With an injection of 1 cc. of prostigmine methylsalicylate the condition of the eyes was brought to almost normal twenty minutes later. The authors consider this test of extreme value in differentiation of congenital ophthalmoplegia and the paralyses of the ocular muscles of any other origin.

They discuss fully the cause, symptoms, diagnosis and treatment of myasthenia gravis.

H. F. CARRASQUILLO.

DIAGNOSIS OF LOCALIZATION OF THE CLAUDE BERNARD–HORNER SYNDROME. R. R. BARRIOS, Arch. de oftal. de Buenos Aires **18:** 629 (Nov.) 1943.

The author states the criteria for localization of the lesion in the sympathetic tract in cases of the Horner syndrome in which no accompanying symptoms make possible such localization.

The lesion producing the condition may be supraganglionic (from the hypothalamus to the anterior horn cells of the cord at the level of the seventh and eighth cervical and the first thoracic vertebrae), preganglionic (from the anterior horn cells to the superior cervical ganglion) and postganglionic (from the superior cervical ganglion to the periphery).

When the lesion is in the first neuron, there are signs of sympathetic excitation in the facial or ocular distribution of the nerve. The miosis is not fixed; some activity of the pupil is brought about by sensitive stimuli coming from below the lesion in the tract. There is a higher response to the dilating action of cocaine than in the unaffected eye.

When the lesion is in the second neuron, there is absence of the mydriatic reaction to sensitive stimuli. The mydriatic response to cocaine does not occur as in the case of lesions of the first neuron. The value of the sign of increased pupillary response (mydriasis) to epinephrine, as stated by other observers, is questioned by the author.

When the lesion takes place in the third neuron, the symptoms of sympathetic paralysis are more pronounced. The response to pilocarpine (increased sweating) in the area of distribution of the nerve is greater. This concept, however, is not definite. An increase in the action of epinephrine when the lesion is in the second neuron is questioned by the author. Barrios has proved experimentally in dogs that acetylcholine produces neuronal excitation (mydriasis and retraction of the nictitating membrane) if the third neuron is intact. The idea is expressed that this test may be made on the human subject. It was found that electric stimulation of the anterior ciliary nerves across the sclera in the anterior segment of the eye produces circumscript mydriasis if the third neuron is intact.

H. F. CARRASQUILLO.

Ocular Muscles

THE FREQUENCY OF SQUINT. M. O. BOYLE, Am. J. Ophth. **27:** 1413 (Dec.) 1944.

Boyle's study of the frequency of squint bears out the generally accepted views.

The incidence of squint in children is usually between 1 and 2 per cent. In school children convergent squint and in adults divergent squint has the higher incidence. As to the time of onset, 1.7 per cent of the cases of convergent squint and 22 per cent of the cases of divergent squint develop after the age of 7 years.

W. ZENTMAYER.

ESSENTIAL REQUIREMENTS FOR A GOOD ORTHOPTIC DEPARTMENT. D. DICKE, Am. J. Ophth. **27:** 1417 (Dec.) 1944.

Dicke stresses the need of cooperation between the ophthalmologist and the orthoptic technician and the necessity for the latter to understand and be interested in children. She discusses treatment, the necessity for minimal fees and the essential equipment required.

W. S. REESE.

CONGENITAL DEFICIENCY OF ABDUCTION OF THE EYES (DUANE'S SYNDROME). A. J. ELLIOT, J. Canad. M. Serv. **1:** 437, 1944.

Elliot reports 3 cases of congenital deficiency in abduction of the eyes. In 1 case a bilateral retraction syndrome was present, while in the other 2 cases a unilateral retraction syndrome was found. A limited review of the literature is given.

The article is illustrated.

W. ZENTMAYER.

Orbit, Eyeball and Accessory Sinuses

EXOPHTHALMOS SECONDARY TO EDEMA AND DEGENERATIVE CHANGES IN ORBITAL TISSUES. M. H. SOLEY, J. Nerv. & Ment. Dis. **99:**865 (May) 1944.

Soley discusses the extreme degree of exophthalmos for which Naffziger recommended orbital decompression. At the thyroid clinic of the University of California a considerable number of patients were encountered in whom exophthalmos was a presenting feature, and yet the majority of these patients had about the same degree of hyperthyroidism, the same sized thyroids and the same levels of oxygen consumption as the average run of patients with thyrotoxicosis in whom exophthalmos was not prominent. The author reviews the history of 37 patients with severe exophthalmos. The symptoms of exophthalmos were "pop eyes," excessive watering, photophobia, blurring of vision, either persistent diplopia or diplopia with ocular fatigue, swelling about the eyes, conjunctival injection, scratchy lids, frontal headache and pain on pressure against the eyeball. The signs were exophthalmos; periorbital and scleral edema; injection of the scleras; widened palpebral fissures; lid lag; frequent winking (probably due to drying and irritation of the globe); impaired convergence, often more pronounced in one eye, and, in the worst cases, engorged retinal veins, papilledema and corneal ulceration. Twenty-five of these patients had thyrotoxicosis at the time the exophthalmos was a presenting complaint. Basal metabolic rate, size of the thyroid, age and other factors did not differ materially from those of corresponding routine thyrotoxic patients. Special interest in the problem of exophthalmos probably accounts for the rather large number of patients in whom severe ocular symptoms were noted early in the course of their disease, before they were treated. The author believes it is important to segregate such patients and to consider the condition of their eyes with special care. If the patient has hyperthyroidism when first seen, roentgen therapy to the thyroid is preferred to subtotal thyroidectomy, in order to allow a more gradual return to a healthy thyroid status and to avoid the complication of myxedema. However, if the goiter is large or nodular, operative treatment is still indicated, and the patient should be reexamined frequently afterward. Indications for orbital decompression are changes in function of extraocular muscles (paresis or complete loss of function), impairment of vision, corneal ulceration or pronounced edema of the conjunctivas or scleras. Because progression of exophthalmos occurs in nearly one half of all thyrotoxic patients who have subtotal thyroidectomy for hyperthyroidism, patients in this category should be watched most carefully after thyroidectomy.

J. A. M. A. (W. ZENTMAYER)

Retina and Optic Nerve

RETRO-BULBAR NEURITIS: FIVE CASES DUE TO PARA-NASAL SINUSITIS. R. FORD, Brit. J. Ophth. **28:** 511 (Oct.) 1944.

Five cases of retrobulbar neuritis supposedly due to paranasal sinusitis are reported. The fact that in the 5 cases vision was recovered after defects lasting as long as two or three, or even twenty-nine years points less to neuritis than to pressure on optic nerve fibers, analogous to that exerted by a pituitary tumor, removal of which even after prolonged pressure on the optic nerve is followed by restored sight. The term "retrobulbar neuritis" is less accurate for the condition than "pressure paresis of the optic nerve." Ford states that it is when the sinusitis is hidden, i. e., when the secretion is retained, that retrobulbar neuritis develops. As soon as it becomes obvious, either spontaneously or through nasal drainage, the retrobulbar neuritis tends to disappear.

W. ZENTMAYER.

ANGIOMA OF THE RETINA. I. C. MICHAELSON, Brit. J. Ophth. **28:** 522 (Oct.) 1944.

A sergeant complained of sudden onset of defective vision in the left eye two weeks previously. Visual acuity with a correction of — 6.50 D. sph. ⊂ — 0.50 D. cyl., axis 180 was 6/36. Examination of the fundus showed a large retinal detachment, chiefly in the lower quadrant, with a tear in the 4 o'clock meridian about 12 mm. from the disk. The inferior temporal artery and vein were broad and tortuous and appeared to end in a raspberry-colored angioma, of about ⅓ disk diameter. General medical examination revealed nothing abnormal.

Micropuncture was made into the tumor, and three micropunctures were made in a vertical line up and in from the tumor with the intention of catching the feeding artery. Surface diathermy with the micropuncture was used. About three months later visual acuity was 6/9, and the visual field had increased in size. The retina was in position; the tumor appeared occluded; the feeding artery was of normal size, and the draining vein could not be definitely traced.

The article is illustrated.

W. ZENTMAYER.

Trachoma

SULFANILAMIDE IN TREATMENT OF TRACHOMA. L. PARADOKSOV, Vestnik oftal. **22:** 41, 1943.

The treatment of trachoma with sulfanilamide has been used since 1940. The dose of sulfanilamide employed was 0.3 cc. given five times a day for ten days, the course being repeated three to five times at intervals of five days. Sulfanilamide powder was also applied locally to the conjunctiva of the everted lids once or twice daily. The last therapeutic measure made the patients comfortable. One hundred and fifty patients, most of whom (140) had trachoma 3, were treated; of these, 112 patients had a complication in the form of pannus or, occasionally, ulcer of the cornea, trichiasis or entropion. In some patients, with trachoma 1 and 2, combined expression of the follicles and chemotherapy were employed.

Two tables and the histories of a few patients with persistent trachoma which did not respond to any therapy except administration of sulfanilamide illustrate the effectiveness of chemotherapy.

Paradoksov comes to the following conclusions: 1. Sulfanilamide is an effective remedy in the treatment of trachoma. 2. The combination of expression, or massage, of the follicles and sulfanilamide therapy frequently gave the best results. 3. It is best to have intervals in the course of treatment of trachoma with sulfanilamide. 4. Administration of sulfanilamide, with use of the powder in the conjunctiva, aids greatly in reduction of the inflammatory process in the cornea (ulcer, pannus) and conjunctiva.

O. SITCHEVSKA.

Vision

TEMPORARY STIMULATION OF EMMETROPIC VISUAL ACUITY. J. E. LEBENSOHN and R. R. SULLIVAN, U. S. Nav. M. Bull. **43:** 1 (July) 1944.

Lebensohn and Sullivan say that visuopsychic excitation seems to be the only factor common to the procedures recommended for improving natural vision. An increase of interest, attention and alertness effects keener interpretation of visuosensory stimuli. Drugs that accelerate cortical or sympathetic activity should be effective. This view is supported by recent experiments on one of the fundamental measurements of visual function, the fusion frequency of flicker, which is indicative of the excitability of the visual system. An increase of acuity above that attainable by a careful refraction would emphasize how much the psychic cortex participates in measurements of visual acuity. Fifty men were selected for study whose visual acuity in each eye was 20/20 or better, naturally or with glasses. Acuity was tested on the double broken circles of the Ferree-Rand chart. From a stopwatch record of the ten letter reading, the speed per letter in tenths of a second was noted and the average of three trials recorded. In every subject the eyes were examined separately and binocularly, so that each examination involved three tests for speed and acuity respectively. Each man was tested at 8 a. m. and was then given a placebo as a control or 10 mg. of amphetamine sulfate or 3 cc. of nikethamide, and the tests were repeated at 10 a. m. and 2 p. m. A consistent improvement in acuity and reading speed was induced by nikethamide and amphetamine in both slow and fast readers. Nikethamide or amphetamine improves reading speed more than visual acuity, but the effect of amphetamine on both visual functions is more pronounced than the effect of nikethamide. Analysis of 150 tests shows that in 44 cases amphetamine was relatively superior to nikethamide in stimulating visual acuity and in 126 cases in increasing reading speed. Amphetamine apparently exerts a more beneficial influence on normal than on ametropic vision. This was proved by comparative tests on 16 persons with ametropia. The authors conclude that the psychogenic origin of many visual complaints is probably insufficiently appreciated. The visuopsychic cortex can be stimulated by various measures, but amphetamine sulfate in small doses (from 5 to 10 mg.) is a simple, safe and efficient agent for this purpose.

J. A. M. A. (W. ZENTMAYER)

Sympathetic Ophthalmia

CHANGES IN THE BLOOD AS A SYMPTOM OF THREATENING SYMPATHETIC OPHTHALMIA: PRELIMINARY REPORT. N. G. RABINOVITCH, Vestnik oftal. **22:** 20, 1943.

The morphologic changes in the blood, particularly the variations in the monocyte count, were observed in 120 cases of severe perforating injury of the eye. In 42 cases the eye was enucleated, and the diagnosis was confirmed by histologic examination; in 78 cases there was severe or mild iridocyclitis. In all but 13 cases in which the eye was enucleated the monocyte count was as high as 8 to 12 per cent. In the second group, of 78 cases, the monocyte count corresponded to the severity of the iridocyclitis, i. e., the count was high with severe iridocyclitis and low with the milder form. In 13 cases in which the eye was enucleated and the histopathalogic diagnosis was endophthalmitis, the monocyte count was fairly normal. The number of monocytes decreased very slowly after enucleation, an indication that the organism was still infected.

Thus, the high monocyte count is a fair indication of threatening sympathetic ophthalmia, and one should be guided by it in deciding to enucleate the injured eye.

O. SITCHEVSKA.

Therapeutics

ROENTGEN THERAPY OF CERTAIN DISEASES OF THE EYE. A. BELTIUKOVA, Sovet. vestnik oftal. **22:** 35, 1943.

Roentgen therapy was used with 51 patients, and the results for 42 of this series were observed and analyzed fully. In 20 patients with trachoma, secondary glaucoma or iridocyclitis roentgen therapy did not give favorable results. The full history and the course of treatment are given for 22 patients with tuberculosis of the anterior segment of the eye. In all these patients the Mantoux reaction was positive, and the possibility of syphilis was eliminated. Many of the patients had received the usual therapeutic measures for many months or years without results. Nineteen such patients suffered from keratoscleritis (9 had sclerosing keratitis) and inflammation of the iris and ciliary body.

A dose of 160 kilovolts and 4 milliamperes was delivered with a filter of 0.5 mm. of copper and 3 mm. of aluminum at a distance of 60 cm., usually every twelve days; this dose was from 10 to 20 per cent of the unit skin dose. As a rule, there was an exacerbation of the process for a few days. Frequently a fresh opacity, possibly a leukocyte reaction, was observed in the cornea near the inflammatory lesion. The best results were obtained in the patients with chronic, severe sclerosing keratitis.

Beltiukova summarizes the results as follows:

1. Roentgen therapy gave excellent results in the treatment of keratoscleritis and keratouveitis. The inflammation subsided; there was no recurrence, and vision was improved, so that the patient could resume his industrial work.

2. When a proper dose of from 10 to 20 per cent of the unit skin dose was employed, administered at intervals of twelve days, the general dose being not higher than 60 per cent of the unit skin dose, no epilation or cataracts were observed.

O. SITCHEVSKA.

Society Transactions

EDITED BY DR. W. L. BENEDICT

COLLEGE OF PHYSICIANS, PHILADELPHIA, SECTION ON OPHTHALMOLOGY

WARREN S. REESE, M.D., *Chairman*

GEORGE F. J. KELLY, M.D., *Clerk*

Dec. 21, 1944

Suprasellar Meningiomas Associated with Scotomatous Field Defects. DR. N. S. SCHLEZINGER, DR. B. J. ALPERS and DR. B. P. WEISS.

In the establishment of a diagnosis of suprasellar tumor, such as a meningioma, the syndrome of optic nerve atrophy and bitemporal field defects has previously been emphasized. In order further to increase efficiency in establishing an early diagnosis of suprasellar meningioma, we believe that the concept of this syndrome should be broadened to include expanding scotomatous field defects in conjunction with atrophy of the optic disks.

Characteristically, the scotomatous field defects become identifiable as a clue to the existence of a chiasmal lesion by uniting with a peripheral encroachment on the visual field at some point so as to produce an expanding sector defect.

Four cases were described which demonstrate the early occurrence of visual loss with scotomas in the visual fields in patients with suprasellar meningiomas. These cases clearly indicate that the syndrome of such scotomatous field defects and optic nerve atrophy always requires exclusion of the diagnostic possibility of a suprasellar tumor before being attributed to retrobulbar neuritis or multiple sclerosis. Headache is often a prominent feature in cases of suprasellar tumor and aids considerably in directing attention toward such a lesion. In the absence of headache and of deformities of the sella turcica, the combination of optic nerve atrophy and either bitemporal or scotomatous field defects is sufficient to warrant the complete examination of the spinal fluid and pneumoencephalographic study.

REPORT OF CASES

CASE 1.—A woman aged 31 was admitted to the hospital on Sept. 16, 1939 with a history of failing vision for eight months, including a period of partial remission. Examination showed bilateral optic nerve atrophy with a central scotoma of the left eye and a combined central and peripheral field defect of the right eye. There was progression of symptoms with severe headaches for the year prior to her death, on April 19, 1942, following operation. The diagnosis of suprasellar meningioma was verified.

CASE 2.—A girl aged 16 was admitted to the hospital on Jan. 18, 1940 with a history of headaches for three years and impairment of vision for four months. Examination showed bilateral optic nerve atrophy with combined central and peripheral field defects of both eyes. Death occurred on Feb. 10, 1940, after operation. The diagnosis of suprasellar meningioma was verified.

CASE 3.—A woman aged 31 was admitted to the hospital on April 24, 1940 with a history of rapidly failing vision of the right eye for four months and of the left eye for one month. Examination showed anosmia on the left side; bilateral optic nerve atrophy, with papilledema in the right eye, and combined central and peripheral field defects of both eyes. There was progression of visual impairment prior to operation, on May 2, 1940. The patient was discharged, with a verified diagnosis of suprasellar meningioma.

CASE 4.—A woman aged 23 was admitted to the hospital on Aug. 19, 1942 with a history of headaches and increasing visual impairment of the left eye for three months, followed by failing vision of the right eye for one month. There was optic nerve atrophy in the left eye, with merged central and inferior altitudinal peripheral defects of that eye, and a central scotoma for color in the right eye. Visual impairment was progressive. The diagnosis of suprasellar meningioma was verified.

DISCUSSION

DR. WALTER I. LILLIE: This excellent presentation should not pass without praise. Acute, precipitous visual changes are due to inflammatory, vascular or toxic conditions, while tumors usually produce a slowly progressive visual syndrome. In the prechiasmal area this does not hold true, owing to the anatomic variations, so well described by Shaeffer and de Schweinitz. Suprasellar meningiomas almost always have their origin in the tuberculum sellae (Cushing), and a certain percentage produce a prechiasmal syndrome. The latter group is illustrated by the authors' cases.

One must decide early whether the lesion is inflammatory or neoplastic. A thorough general and neurologic examination, including an examination of the spinal fluid, is important before a definite diagnosis is made. Multiple sclerosis is a common cause of the prechiasmal syndrome, and the spinal fluid usually shows an increase in the number of cells and a colloidal gold curve of zone 1 type. With a chronic type of inflammation—chronic basal arachnoiditis—or with suprasellar tumors metabolic changes occur at the same time as or after the visual disturbances, a point which easily differentiates these lesions from the intrasellar type. The encephalogram is

also an aid in differentiating the suprasellar tumor and chronic basal arachnoiditis.

These two conditions are surgical and do not respond to medical therapy. Ophthalmologists should insist on an early diagnosis, so that the neurosurgeon can institute the proper therapy. The results are excellent; the mortality rate is low, and the patient obtains useful vision.

DR. N. S. SCHLEZINGER: Dr. Lillie has given an excellent discussion of the paper and has emphasized the points which needed stressing.

Penetration of Penicillin in Rabbit Eyes with Normal, Inflamed and Abraded Corneas.
DR. IRVING H. LEOPOLD and (by invitation) DR. WILLIAM O. LAMOTTE JR.

This paper was published in full in the January 1945 issue of the ARCHIVES, page 43.

DISCUSSION

DR. FRANCIS HEED ADLER: It is not often that the simplest way of treating a disease turns out to be the best, but I believe that these experiments show this to be true in the case of penicillin therapy of acute inflammations of the anterior segment. I have seen sensitivity to penicillin develop in 2 patients when the drug was used locally. I should like to ask Dr. Leopold whether he observed sensitization in experimental animals. I should also like to ask whether the organism that he used to obtain experimental keratitis was one which penicillin itself would kill. If so, did he find any difference in the curative value of penicillin with different methods of administration?

DR. IRVING H. LEOPOLD: In answer to Dr. Adler's question concerning the development of local sensitivity to the use of penicillin, it is surprising how few cases of sensitization one sees, considering the huge doses of penicillin that have been used systemically, as well as locally. Actually, however, there are reports appearing in the literature which suggest local sensitization to penicillin. Two cases of contact dermatitis due to penicillin have been reported (Binkley, G. W., and Brockinole, A.: Dermatitis from Penicillin, *Arch. Dermat. & Syph.* **33**:326 [Nov] 1944). In the same issue (page 330), Rostenberg refers to a report by Welch and himself that penicillin when injected intradermally will produce sensitivity. Physicians must be on the lookout for allergic manifestations of this drug, as they have been for the sulfonamide compounds. Actually, we have seen no signs of contact dermatitis in the rabbits, but the rabbit is a notoriously poor animal in which to demonstrate such hypersensitivity.

In answer to the second question, this organism, Pasteurella lepiseptica, was obtained by routine culture of material from our rabbits' eyes. It is a gram-negative bacillus, which we found to be sensitive to penicillin. Bacteriologic studies made us think at first that it was a previously undescribed organism, but further study and consultation with Dr. L. A. Julianelle and Dr. L. F. Rettger revealed that the bacillus, although not identical with, most closely resembled Past. lepiseptica. We produced corneal infection with this organism in a series of rabbits and compared the therapeutic effectiveness of local and systemic administration of penicillin. The intramuscularly administered penicillin failed to have any influence on the corneal ulcers, whereas all eyes treated locally with penicillin responded well to the therapy.

DR. FRANCIS HEED ADLER: Then the weight of evidence would point toward local use of the drug in all cases? What evidence there is suggests this method, rather than intramuscular injection.

DR. IRVING H. LEOPOLD: Yes, all studies indicate the superiority of local over systemic administration of penicillin for corneal infections due to penicillin-sensitive organisms.

Syphilitic Iritis, with Particular Reference to the Herxheimer Reaction as a Diagnostic Aid and Response to Different Methods of Treatment, Including Penicillin Therapy.
DR. JOSEPH V. KLAUDER (by invitation) and DR. GEORGE J. DUBLIN.

How valid is the diagnosis of syphilitic iritis when based solely on a positive Wassermann reaction or when made in the late stage of syphilis or in cases in which the duration of infection is unknown? What are the criteria of the efficacy of antisyphilitic treatment in justifying the conclusion that the iritis is due to syphilis? Antisyphilitic treatment exerts a nonspecific effect on iritis. Judgment at times is difficult, since both local treatment and nonspecific therapy exert a favorable action on iritis. The purpose of this paper was to discuss these questions, as well as the Herxheimer reaction of the ocular lesion as evidence of the syphilitic causation. The intensification of the inflammatory process (constituting the Herxheimer reaction) was evaluated by examinations with the slit lamp conducted before and soon after antisyphilitic treatment. The Herxheimer reaction, as observed through the corneal microscope, had not previously been employed in diagnosis.

Of 33 patients with syphilitic iritis, 3 were treated with penicillin. The case record of 1 of these 3 patients was given in detail; the flare-up of the ocular lesion (Herxheimer reaction) after treatment with penicillin was described, and the period required for the iritis to become quiescent and the effect on the lesions of the skin and the mucous membranes were discussed.

In order to avoid too pronounced a Herxheimer reaction and too rapid regression of the

inflammatory lesion (therapeutic paradox), reduced initial doses of penicillin were employed—10,000 units in each of the first four injections, as compared with 50,000 units employed in treatment of early syphilis. A total dose of 2,400,000 units of penicillin was administered to each of 2 patients, and 1,200,000 units of penicillin was administered in treatment of the third patient, who had syphilitic iritis and associated secondary syphilis.

The iritis of the 3 patients treated with penicillin became quiescent within twelve days, as compared with two to five weeks required for patients given chemotherapy or fever treatment combined with chemotherapy.

DISCUSSION

DR. ALFRED COWAN: I agree with the authors that there are few objective features in syphilitic iritis or uveitis that cannot be found in almost any other type of severe uveitis. In my experience papules are rare. Certainly, ophthalmologists seldom make any distinction between the two forms of iritis, so that to consider some sign as characteristic of syphilitic uveitis is pretty far fetched.

Formerly, a diagnosis of syphilitic iritis was much more readily made than it is today. Then, severe iritis, if the patient was not alcoholic and did not have tuberculosis or rheumatism, was regarded of syphilitic origin. That explains the earlier large proportion of cases of iritis in which syphilis was supposed to be the cause, as compared with the present number of cases of syphilitic iritis.

In how many of the 33 patients did Dr. Dublin find lesions resembling papules? I recall that in cases in which the iritis was thought to be syphilitic the ophthalmologist was careful about sending the patient to the syphilologist before first treating him, lest the treatment should be too severe or by a severe reaction should produce damage that could never be repaired.

DR. GEORGE F. J. KELLY: Did these patients show general signs of a Herxheimer reaction, or was this response confined to the eyes? It was stated that the eyes of these patients were examined with the corneal microscope sixteen to eighteen hours after the injection of penicillin. Were they examined before receiving the injections? Dr. Dublin referred to penicillin as a spirocheticide. Does he regard that as proved?

DR. GEORGE J. DUBLIN: In reply to Dr. Cowan's question whether we saw any nodules in the iris in our series of patients: It is my impression that such lesions were noted in 5 patients. The nodules were distinct masses and were not noted at the first examination. The last patient, who had a large mass in the iris at the time I saw him and described the case to my resident at Wills Hospital, was examined by Dr. Cowan three days later.

DR. DUBLIN: Did Dr. Cowan fail to see any nodule? Dr. Klauder referred to this patient as showing tremendous improvement with fever therapy prior to treatment with penicillin. On my first examination, I noted a pronounced massive nodule in the lower portion of the iris below the pupillary border. Five days later there was no evidence of this mass. Slight atrophy of the iris was present at this point. The mass disappeared faster than any that I have ever observed.

In regard to the question whether we noted any general manifestations of too rapid a Herxheimer reaction, it is my belief that such a response occurred only once—in a patient who complained of severe headaches after injection of penicillin. A patient is usually examined with a corneal microscope one or two hours prior to injection of an arsenical and approximately sixteen to eighteen hours after the injection. From 0.30 to 0.45 Gm. of neoarsphenamine is given in an attempt to avoid the possibility of too rapid destruction of the tissues or too rapid regression of the lesion.

It is interesting to note the different features of syphilitic iritis with the slit lamp microscope, for this is the first time we have been fortunate enough to be able to see, with a fairly high degree of clearness, pathologic lesions in the anterior segment of the eye. We have divided the Herxheimer reaction into four stages: In the first stage, after eighteen hours, there are some clouding of the cornea, some fine keratitic precipitates and a slight aqueous flare, and in the second stage, pronounced haziness and cloudiness of the cornea, a pronounced aqueous flare with turbidity and many floating cells in the aqueous. In the third stage, all the aforementioned changes are present, together with nodules in the iris. Some of the nodules have a reddish tint, while others are yellowish. All the nodules we have noted have been close to the pupillary border or immediately adjacent to it. In only 1 case was a mass noted near the root of the iris. In the fourth stage there is a spongy exudate (fibrinous iritis) with a "frozen" aqueous.

Prior to the advent of the slit lamp evidence of a Herxheimer reaction was deduced from the appearance of various physical signs, such as headache following injection of an arsenical or rupture of an aneurysm after antisyphilitic treatment. We do not feel that the Herxheimer reaction, particularly as noted with the slit lamp microscope, is an infallible sign of syphilis. We believe, however, that it is of diagnostic aid in a high percentage of cases and is an addition to the evidence obtained by laboratory and clinical methods of examination, including the Wassermann test. Even when there is distinct clinical evidence of syphilis, the Wassermann reaction may be negative. We have had a small percentage of Herxheimer reactions in cases of nonsyphilitic

iritis, but we are unable to explain them at this time. We are working on a number of control cases, however, and we hope to have more accurate data and to reach more definite conclusions in the near future. We do feel, however, that a positive Herxheimer reaction as noted with the slit lamp microscope is an addition to the evidence obtained by laboratory tests.

WARREN S. REESE, M.D., *Chairman*

GEORGE F. J. KELLY, M.D., *Clerk*

Jan. 18, 1945

Acquired Ptosis: Classification and Correction. Dr. EDMUND B. SPAETH.

The author presented an analysis of over 200 cases in which acquired ptosis was either the sole complaint or part of the symptom complex of the underlying condition. From the data a classification based on anatomic and etiologic characteristics was developed. Cases illustrating each division were shown with lantern slides.

The varied surgical means for correcting these conditions were presented. All forms of symptomatic ptosis are not surgical conditions. Those which are need individual attention, depending on the underlying factors.

This paper will be published in the *American Journal of Ophthalmology*.

DISCUSSION

DR. JAMES S. SHIPMAN: Dr. Spaeth has shown many forms of ptosis that many did not know occurred. Usually one thinks of ptosis as a congenital condition, which can be corrected by one of many different operations; the reason there are so many operations for ptosis is that none of them is perfect.

Dr. Spaeth has demonstrated that ptosis does occur after birth, as the result of many factors, and that it is by no means always congenital. Before one operates on any patient for ptosis, one should first determine whether or not the condition is congenital. If it is a case of acquired ptosis, what is the cause? What is the pathologic condition? Unless these factors are known, one may get into difficulties that could have been avoided. Certainly, in cases of ptosis due to syphilis or myasthenia gravis, one does not try operative intervention.

As Dr. Spaeth has shown, the operative principle in correction of acquired ptosis is much the same as that used in correction of the congenital form. The cause, the amount of scar tissue and the pathologic process, however, modify a great deal the technic that is to be followed.

Aberrations of the Eye. DR. ALFRED COWAN.

Thomas Young, as early as 1801, showed conclusively that the eye is neither achromatic nor aplanatic. Many others, particularly Volkmann, Donders, J. H. Knapp and Tscherning, investigated the aberrations of the eye; but the most thorough study of the actual image of the eye was made by Gullstrand. He found that the caustic surface is extremely intricate and that it varies as the meridional section is revolved around the axis.

In the eye one has not only all the aberrations which must be eliminated in a well designed artificial optical instrument but the added faults that exist in a living asymmetric organ.

The aberrations of the eye are physiologic, and their absence would be of no use to the eye. The effect of the circle of least confusion is that of a point image as long as its diameter does not exceed the resolving power of the eye. The retina is sensitive to distinct visual acuity only in a very small area at the fovea, and for this reason only that part of the caustic which is found most useful is utilized. The surrounding halo is disregarded. Also, because of the peculiar structure of the retina, the effects of such factors as marginal astigmatism, curvature of field and distortion are negligible. The faults of the optical system of the eye do not interfere with distinct visual acuity.

DISCUSSION

DR. FRANCIS HEED ADLER: This is a valuable paper, and it indicates fair mindedness in at least one physiologic opticist. The majority seem to have such faith in geometric optics that they are like an economist who wilfully blinds himself to the frailties of human nature in order to believe wholeheartedly in the doctrines of Karl Marx. They insist on speaking of point images on the retina, although they know they do not exist. This has seriously retarded knowledge of visual physiology, and Dr. Lancaster is quite right in emphasizing the importance of learning about image formation in the eye as it occurs in nature, and not as it is pictured in geometric optics. I agree with Dr. Cowan that geometric optics should be taught as the basis for physiologic optics, but the student must be carried beyond this and instructed in image formation as it actually occurs in the human eye.

DR. I. S. TASSMAN: I should like to commend Dr. Cowan for his excellent presentation and for pointing out the value of retinoscopy in at least obtaining an approximate determination of the refractive state.

It seems to me that in recent years there has been a tendency to overemphasize subjective examination in refraction, or at least to emphasize certain subjective tests and methods for determining the result in refraction. At the same time, in a great many instances, the objective examina-

tion is neglected. A paper of this kind will help to overcome some of the objections that have been raised to objective examination and retinoscopy, particularly in cases in which cycloplegics are employed. One of the chief objections to the use of cycloplegics has been the very large pupil and the difficulty in making a retinoscopic examination of the central area.

This, as Dr. Cowan pointed out, should present no difficulty to one with any experience in retinoscopy. I should go a step further and add that retinoscopic examinations should be performed in every case preliminary to subjective study regardless of whether or not a cycloplegic has been employed. I think it is a mistake that, in the examination of presbyopes especially, retinoscopic examination is often omitted.

Infection of the Human Eye with Torula Histolytica. Dr. Charles Weiss, Dr. M. C. Shevky and Dr. I. H. Perry.

Through the cooperation of Dr. Martin I. Green, of San Francisco, we examined material obtained by trephining the sclera of a patient for whom the clinical diagnosis of a possible cyst of the retina had been made. The history is briefly as follows:

A., a white man aged 56, had been in an automobile accident in 1941. In July 1943 Dr. R. J. Nutting, of Oakland, Calif., discovered a growth in his right eye and enucleated it in October 1943. Three months later the patient complained of poor vision in the left eye. He soon manifested mental symptoms and was confined to a sanatorium. The eye showed fine deposits on the posterior surface of the cornea and on the anterior surface of the lens. There were "floaters" in the aqueous and the vitreous. A diagnosis of "possible cyst of the retina with retinal detachment and uveitis" was made. The sclera was trephined, and material obtained from culture showed the presence of a yeastlike organism which we identified as Torula histolytica (Cryptococcus hominis). This is the second case of an ocular infection due to T. histolytica to be reported in the literature.

In its cultural characteristics the organism resembled the torula of Benham and Hopkins' group III when grown in corn meal agar. Its ability to ferment carbohydrates was weak and variable. No gas was formed. Gelatin was not liquefied. There was abundant budding, but neither myceliums nor endospores were formed. The organism killed mice when injected intraperitoneally or intravenously.

When the culture was injected into the anterior chamber of the eyes of rabbits, it was possible to observe early pathologic changes. The inferior part of the anterior chamber became opaque, and a pannus developed on the surface of the cornea.

Histologically, there was a slight exudate consisting of polymorphonuclear cells and some monocytes along the periphery of the cornea. Along the anterior surface of the iris and the posterior surface of the cornea was a delicate festoon of rosettes. In the center of each rosette was a torula, surrounded by a single row of cells (polymorphonuclear cells and monocytes). Torulas were seen engulfed in macrophages.

DISCUSSION

Dr. William O. LaMotte Jr.: This organism, known as T. histolytica, was brought to the attention of my colleagues and myself in the department of ophthalmology of the University Hospital during the past year through an opportunity of observing the eyegrounds of a patient in the orthopedic-pediatric services who subsequently died of an infection with T. histolytica of such widespread degree that the term torulosis could aptly be applied to it. A report of this case will be published soon by Dr. Joseph Snyder (Meningitis Due to Infection with Torula Histolytica: Report of a Case, *Arch. Neurol. & Psychiat.* **53:** 392 [May] 1945) resident pathologist at the University Hospital. The patient, a 6 year old girl, was originally admitted for treatment of chronic osteomyelitis of the left femur and right humerus with penicillin. The offending organism was hemolytic Staphylococcus aureus. It was not long after her admission that signs of a generalized systemic infection and involvement of the central nervous system appeared, including an increase in the cerebrospinal fluid pressure. Many torulas were isolated from the spinal fluid, both on direct smear and by culture. Despite massive doses of penicillin, given both intramuscularly and intrathecally, the child died. The penicillin therapy did not influence the number of torulas in the spinal fluid. It was about six weeks after the first sign of involvement of the central nervous system that the child died. During all this time we had the fundus under observation. There were no definite changes until a week before her death, at which time bilateral papilledema of 2 to 3 D. appeared. Also, in the region of the posterior pole of each eye were a number of poorly defined, pale yellow areas, of about 1/8 disk diameter and deep to the retinal vessels, having somewhat the appearance of deep necrosis or thick exudates.

At autopsy the orbits were unroofed and the posterior segments of the eyes removed. In view of the fact that the torula was found histologically in practically every tissue of the body, it was felt that the presence of torulas might explain the patches observed ophthalmoscopically in the eye before death. Sections did show a few nests of the organism in the retina, although not in as great abundance as we expected, judging from the lesions seen ophthalmoscopically.

I should like to point out that in all the articles that have appeared, and in the recent one by Dr. Martin Cohen (Binocular Papilledema in Case of Torulosis Associated with Hodgkin's Disease, ARCH. OPHTH. **30:** 477 [Dec] 1944), the statement is made that the genus Torula is widespread in nature. I understand from Dr. Weidman, however, that an organism which can produce infection both in man and in animals has not been found in nature, that the organisms of this group which are widespread are all saprophytic and that, in his opinion, it is still problematic where the particular organism which causes human infection resides between the appearance of these relatively infrequently reported cases. I was wondering whether in his experiments Dr. Weiss used only the organism isolated from his patient or whether he actually attempted to produce animal infection with some of the strains of Torula which have been found widespread in nature. Dr. Weiss's presentation is instructive; perhaps in the near future, one will be able to make use of diagnostic tests.

DR. CHARLES WEISS: We used only the strain which we isolated from the patient to produce the lesions in the rabbit's eye.

News and Notes

EDITED BY DR. W. L. BENEDICT

GENERAL NEWS

Canadian Ophthalmological Society.—The seventh annual meeting will be held in Montreal, Canada, at the Montreal Neurological Institute, 3801 University Street, on Friday, June 15, 1945.

Thursday, June 14.—Council meeting and dinner at the University Club of Montreal, 2047 Mansfield Street at 7 p. m.

Friday, June 15.—Executive Session: 9 a. m.

Scientific Sessions: The following program will be presented: 10:30 a. m. Dr. F. A. Aylesworth: A Study of Causes of Blindness in Over 12,000 Cases in Canada; Dr. J. A. MacMillan: The Pathology of Retinal Changes in Tay-Sachs Disease; Dr. J. E. Pelletier: Visual Fields with Tangent Screen; Dr. A. Lloyd Morgan: Recession of the Inferior Oblique.

2:30 p. m. Dr. W. Gordon M. Byers: O'Connor Advancement (Cinch) Operation; First Lieutenant Leo S. Kirschberg, R.C.A.F. (by invitation): Interrelationship of Heterophoria, Stereopsis and Flying Performance in Flying Training; Second Lieutenant J. C. McCulloch, R.C.A.F. (by invitation): Clinical Aspects of Stereopsis; Dr. Charles E. Davies: Orthoptic Treatment in Convergence Insufficiency.

American Orthoptic Council: Examinations for Technicians.—The next examinations by the American Orthoptic Council will be held in September and October 1945.

The written examinations will be held at various cities in the country on Friday, September 7. Only candidates passing the written examinations will be permitted to take the oral and practical tests, to be given in Chicago, Saturday, October 6.

Applications on official forms must be received before July 1, 1945.

Address: The American Orthoptic Council, 23 East Seventy-Ninth Street, New York 21.

American Board of Ophthalmology.—An examination will be held by the American Board of Ophthalmology in Los Angeles in January 1946, at the time of the mid-winter course. Applications for this examination must be filed before September 1. Further information may be procured from the secretary, Dr. S. Judd Beach, Cape Cottage, Maine.

SOCIETY NEWS

Central Illinois Society of Ophthalmology and Otolaryngology.—The first meeting of the Central Illinois Society of Ophthalmology and Otolaryngology was held at Bloomington, Ill., April 21 and 22 at the Gailey Eye Clinic. The program consisted of papers on "Normal Action of Extraocular Muscles," "Lesions of the Esophagus," "Relationship of Muscle Imbalances and Refraction, Principles of Ocular Muscle Surgery and Ophthalmic Plastic Procedures" and "Cancer of Larynx and Lateral Sinus Thrombosis."

The officers of the Central Illinois Society will be listed in the "Directory of Ophthalmologic Societies."

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Association for Prevention of Blindness

President: Dr. P. Bailliart, 66 Boulevard Saint-Michel, Paris, 6e, France.
Secretary-General: Prof. M. Van Duyse, Université de Gand, Gand, Prov. Ostflandern, Belgium.
All correspondence should be addressed to the Secretariat, 66 Boulevard Saint-Michel, Paris, 6e, France.

International Ophthalmologic Congress

President: Prof. Nordenson, Serafimerlasarettet, Stockholm, Sweden.
Secretary: Dr. Ehlers, Jerbanenegade 41, Copenhagen, Denmark.

International Organization Against Trachoma

President: Dr. A. F. MacCallan, 17 Horseferry Rd., London, England.

Pan-American Congress of Ophthalmology

President: Dr. Harry S. Gradle, 58 E. Washington St., Chicago.
Executive Secretaries: Dr. Conrad Berens, 35 E. 70th St., New York. Dr. M. E. Alvaro, 1511 Rua Consolacão, São Paulo, Brazil.

FOREIGN

All-India Ophthalmological Society

President: Dr. B. K. Narayan Rao, Minto Ophthalmic Hospital, Bangalore.
Secretary: Dr. G. Zachariah, Flitcham, Marshall's Rd., Madras.

British Medical Association, Section on Ophthalmology

President: Dr. W. Clark Souter, 9 Albyn Pl., Aberdeen, Scotland.
Secretary: Dr. Frederick Ridley, 12 Wimpole St., London, W. 1.

Chengtu Ophthalmological Society

President: Dr. Eugene Chan.
Secretary: Dr. K. S. Sun.
Place: Eye, Ear, Nose and Throat Hospital, Chengtu, China.

Chinese Ophthalmology Society

President: Dr. C. H. Chou, 363 Avenue Haig, Shanghai.
Secretary: Dr. F. S. Tsang, 221 Foochow Rd., Shanghai.

Chinese Ophthalmological Society of Peiping

President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.
Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen Chienmeng, Peiping.
Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

Faculty of Ophthalmologists

President: Brig. Sir Stewart Duke-Elder, 63 Harley St., London, W. 1, England.
Secretary: Mr. Frank W. Law, 45 Lincoln's Inn Fields, London, W. C. 2., England.

German Ophthalmological Society

President: Prof. W. Lohlein, Berlin.
Secretary: Prof. E. Engelking, Heidelberg.

Hungarian Ophthalmological Society

President: Prof. I. Imre, Budapest.
Assistant Secretary: Dr. Stephen de Grósz, University Eye Hospital, Máriautca 39, Budapest.
All correspondence should be addressed to the Assistant Secretary.

Midland Ophthalmological Society

President: Dr. W. Niccol, 4 College Green, Gloucester, England.
Secretary: Mr. T. Harrison Butler, 61 Newhall St., Birmingham 3, England.
Place: Birmingham and Midland Eye Hospital.

North of England Ophthalmological Society

President: Mr. John Foster, 45 Park Sq., Leeds.
Secretary: Mr. William M. Muirhead, 70 Upper Hanover St., Sheffield.
Place: Manchester, Bradford, Leeds, Newcastle-upon-Tyne, Liverpool and Sheffield, in rotation. Time: October to April.

Ophthalmological Society of Australia

President: Dr. A. James Flynn, 135 Macquarie St., Sydney.
Secretary: Dr. D. Williams, 193 Macquarie St., Sydney.

Ophthalmological Society of Egypt

President: Prof. Dr. Mohammed Mahfouz Bey, Government Hospital, Alexandria.
Secretary: Dr. Mohammed Khalil, 4 Baehler St., Cairo.
All correspondence should be addressed to the secretary, Dr. Mohammed Khalil.

Ophthalmological Society of South Africa

President: Dr. A. W. Sichel, National Mutual Bldg., Church Square, Cape Town.
Secretary: Dr. J. K. de Kock, Groote Kerk Bldg., 32 Parliament St., Cape Town.

Ophthalmological Society of the United Kingdom

President: Mr. Charles B. Goulden, 89 Harley St., London.
Secretary: Mr. Frank W. Law, 30 Devonshire Pl., London, W. 1.

Ophthalmology Society of Bombay

President: Dr. D. D. Sathaye, 127 Girgaum Rd., Bombay 4, India.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14, India.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

Oxford Ophthalmological Congress

Master: Mr. P. G. Doyne, 60 Queen Anne St., London, W. 1, England.
Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.

Palestine Ophthalmological Society

President: Dr. Arieh Feigenbaum, Abyssinian St. 15, Jerusalem.
Secretary: Dr. E. Sinai, Tel Aviv.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and keep it up to date.

POLISH OPHTHALMOLOGICAL SOCIETY

President: Dr. W. Kapuściński, 2 Waly Batorego, Poznań.
Secretary: Dr. J. Sobański, Lindley'a 4, Warsaw.
Place: Lindley'a 4, Warsaw.

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

President: Col. F. A. Juler, 96 Harley St., London, W. 1, England.
Secretary: Dr. Harold Ridley, 60 Queen Anne St., London, W. 1, England.

SÃO PAULO SOCIETY OF OPHTHALMOLOGY

President: Prof. Moacyr E. Alvaro, Consolação 1151, São Paulo, Brazil.
Secretary: Dr. Silvio de Almeida Toledo, Enfermaria Santo Luzia, Santa Casa de Misericordia, Cesario Motta, St. 112, São Paulo, Brazil.

SCOTTISH OPHTHALMOLOGICAL CLUB

President: Dr. S. Spence Meighan, 13 Woodside Pl., Glasgow, C. 3.
Secretary: Dr. Alexander Garrow, 15 Woodside Pl., Glasgow, C. 3.
Place: Edinburgh and Glasgow, in rotation.

SOCIEDAD ARGENTINA DE OFTALMOLOGIA

Chairman: Dr. Jorge Malbrán, Buenos Aires.
Secretary: Dr. Benito Just Tiscornia, Santa Fe 1171, Buenos Aires.

SOCIEDAD OFTALMOLOGIA DEL LITORAL, ROSARIO (ARGENTINA)

President: Prof. Dr. Carlos Weskamp, Laprida 1159, Rosario.
Secretary: Dr. Arturo Etchemendigaray, Villa Constitución, Santa Fé.
Place: Rosario. Time: Last Saturday of every month, April to November. All correspondence should be addressed to the President.

SOCIEDADE DE OFTALMOLOGIA DE MINAS GERAIS

President: Prof. Hilton Rocha, Rua Rio de Janeiro 2251, Bello Horizonte, Minas Geraes, Brazil.
Secretary: Dr. Ennio Coscarelli, Rua Aimorés 1697, Bello Horizonte, Minas Geraes, Brazil.

SOCIEDADE DE OFTALMOLOGIA E OTORRINOLARINGOLOGIA DE RIO GRANDE DO SUL

President: Dr. Luiz Assumpção Osorio, Edificio Vera Cruz, Apartamento 134, Porto Alegre, Rio Grande do Sul.
Secretary: Dr. Fernando Voges Alves, Caixa Postal 928, Porto Alegre, Rio Grande do Sul.

SOCIEDADE DE OPHTHALMOLOGIA E OTO-RHINO-LARYNGOLOGIA DA BAHIA

President: Dr. Theonilo Amorim, Barra Avenida, Bahia, Brazil.
Secretary: Dr. Adroaldo de Alencar, Brazil.
All correspondence should be addressed to the President.

SOCIETÀ OFTALMOLOGICA ITALIANA

President: Prof. Dott. Giuseppe Ovio, Ophthalmological Clinic, University of Rome, Rome.
Secretary: Prof. Dott. Epimaco Leonardi, Via del Gianicolo, 1, Rome.

SOCIÉTÉ FRANÇAISE D'OPHTALMOLOGIE

Secretary: Dr. René Onfray, 6 Avenue de la Motte Picquet, Paris, 7e.

SOCIETY OF SWEDISH OPHTHALMOLOGISTS

President: Prof. K. G. Ploman, Stockholm.
Secretary: Dr. K. O. Granström, Södermalmstorg 4 III tr., Stockholm, Sö.

TEL AVIV OPHTHALMOLOGICAL SOCIETY

President: Dr. D. Arieh-Friedman, 96 Allenby St., Tel Aviv, Palestine.
Secretary: Dr. Sadger Max, 9 Bialik St., Tel Aviv, Palestine.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Frederick C. Cordes, 384 Post St., San Francisco.
Secretary: Dr. R. J. Masters, 23 E. Ohio St., Indianapolis.
In compliance with the request of the Office of Defense Transportation and in the interest of the national war effort a meeting will not be held in 1945.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY, SECTION ON OPHTHALMOLOGY

President: Dr. Gordon B. New, Mayo Clinic, Rochester, Minn.
President-Elect: Dr. Alan C. Woods, Johns Hopkins Hospital, Baltimore 5.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100–1st Ave. Bldg., Rochester, Minn.

AMERICAN OPHTHALMOLOGICAL SOCIETY

President: Dr. S. Judd Beach, 704 Congress St., Portland, Maine.
Secretary-Treasurer: Dr. Walter S. Atkinson, 129 Clinton St., Watertown, N. Y.

ASSOCIATION FOR RESEARCH IN OPHTHALMOLOGY, INC.

Chairman: Dr. Conrad Berens, 35 E. 70th St., New York.
Secretary-Treasurer: Major Brittain F. Payne, School of Aviation Medicine, Randolph Field, Texas.
Assistant Secretary-Treasurer: Dr. Hunter Romaine, 35 E. 70th St., New York.

CANADIAN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY

President: Dr. Alexander E. MacDonald, 170 St. George St., Toronto.
Secretary-Treasurer: Dr. L. J. Sebert, 170 St. George St., Toronto.

CANADIAN OPHTHALMOLOGICAL SOCIETY

President: Dr. R. Evatt Mathers, 34½ Morris St., Halifax, N. S.
Secretary-Treasurer: Dr. Kenneth B. Johnston, Suite 1, 1509 Sherbrooke St. W., Montreal.

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS

President: Mr. Mason H. Bigelow, 1790 Broadway, New York.
Secretary: Miss Regina E. Schneider, 1790 Broadway, New York.
Executive Director: Mrs. Eleanor Brown Merrill, 1790 Broadway, New York.

SECTIONAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. N. Zwaifler, 46 Wilbur Ave., Newark.
Secretary: Dr. William F. Keim Jr., 25 Roseville Ave., Newark.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Watson Gailey, 1000 N. Main St., Bloomington, Ill.
Secretary-Treasurer: Dr. William F. Hubble, 861-867 Citizens Bldg., Decatur, Ill.

CENTRAL WISCONSIN SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. L. J. Friend, 425 E. Grand Ave., Beloit, Wis.
Secretary: Dr. G. L. McCormick, 626 S. Central Ave., Marshfield.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. Paul A. Chandler, 5 Bay State Rd., Boston.
Secretary-Treasurer: Dr. Merrill J. King, 264 Beacon St., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. D. H. O'Rourke, 1612 Tremont Pl., Denver.
Secretary-Treasurer: Dr. C. Allen Dickey, 450 Sutter St., San Francisco.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. James H. Mathews, 1317 Marion St., Seattle, Wash.
Secretary-Treasurer: Dr. Barton E. Peden, 301 Stimson Bldg., Seattle 1.
Place: Seattle or Tacoma, Wash. Time: Third Tuesday of each month except June, July and August.

ROCK RIVER VALLEY EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. J. Sheldon Clark, 27 E. Stephenson St., Freeport, Ill.
Secretary-Treasurer: Dr. Harry R. Warner, 321 W. State St., Rockford, Ill.
Place: Rockford, Ill., or Janesville or Beloit, Wis. Time: Third Tuesday of each month from October to April, inclusive.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. L. D. Gomon, 308 Eddy Bldg., Saginaw, Mich.
Secretary-Treasurer: Dr. Harold H. Heuser, 207 Davidson Bldg., Bay City, Mich.
Place: Saginaw or Bay City, Mich. Time: Second Tuesday of each month, except July, August and September.

SIOUX VALLEY EYE AND EAR ACADEMY

President: Dr. J. C. Decker, 515 Francis Bldg., Sioux City, Iowa.
Secretary-Treasurer: Dr. J. E. Dvorak, 408 Davidson Bldg., Sioux City, Iowa.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. John H. Burleson, 414 Navarro St., San Antonio, Texas.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville, S. C.

SOUTHWESTERN ACADEMY OF EYE, EAR, NOSE AND THROAT

President: Dr. H. L. Brehmer, 221 W. Central Ave., Albuquerque, N. Mex.
Secretary: Dr. A. E. Cruthirds, 1011 Professional Bldg., Phoenix, Ariz.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. W. M. Dodge, 716 First National Bank Bldg., Battle Creek.
Secretary-Treasurer: Dr. Kenneth Lowe, 25 W. Michigan Ave., Battle Creek.
Time: Last Thursday of September, October, November, March, April and May.

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Ray Parker, 218 Franklin St., Johnston, Pa.
Secretary-Treasurer: Dr. J. McClure Tyson, Deposit Nat'l Bank Bldg., DuBois.

STATE

ARKANSAS STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. Raymond C. Cook, 701 Main St., Little Rock.
Secretary: Dr. K. W. Cosgrove, Urquhart Bldg., Little Rock.

COLORADO OPHTHALMOLOGICAL SOCIETY

President: Dr. C. A. Ringle, 912-9th Ave., Greeley.
Secretary: Dr. W. A. Ohmart, 1102 Republic Bldg., Denver.
Place: University Club, Denver. Time: 7:30 p. m., third Saturday of each month, October to May, inclusive.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. F. L. Phillips, 405 Temple St., New Haven.
Secretary-Treasurer: Dr. W. H. Turnley, 1 Atlantic St., Stamford, Conn.

EYE, EAR, NOSE AND THROAT CLUB OF GEORGIA

President: William O. Martin Jr., Doctors Bldg., Atlanta.
Secretary-Treasurer: Dr. C. K. McLaughlin, 526 Walton St., Macon.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. F. McK. Ruby, Union City.
Secretary: Dr. Edwin W. Dyar Jr., 23 E. Ohio St., Indianapolis.
Place: French Lick. Time: First Wednesday in April.

Iowa Academy of Ophthalmology and Oto-Laryngology

President: Dr. J. K. Von Lackum, 117–3d St. S. E., Cedar Rapids.
Secretary-Treasurer: Dr. B. M. Merkel, 604 Locust St., Des Moines.

Kansas State Medical Society, Section on Ophthalmology and Otolaryngology

President: Dr. W. D. Pittman, Pratt.
Secretary: Dr. Louis R. Haas, 902 N. Broadway, Pittsburg.

Louisiana-Mississippi Ophthalmological and Otolaryngological Society

President: Dr. Val. H. Fuchs, 200 Carondelet St., New Orleans.
Secretary-Treasurer: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

Michigan State Medical Society, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Robert H. Fraser, 25 W. Michigan Ave., Battle Creek.
Secretary: Dr. R. G. Laird, 114 Fulton St., Grand Rapids.

Minnesota Academy of Ophthalmology and Otolaryngology

President: Dr. George E. McGeary, 920 Medical Arts Bldg., Minneapolis.
Secretary: Dr. William A. Kennedy, 372 St. Peter St., St. Paul.
Time: Second Friday of each month from October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. William Morrison, 208 N. Broadway, Billings.
Secretary: Dr. Fritz D. Hurd, 309 Medical Arts Bldg., Great Falls.

Nebraska Academy of Ophthalmology and Otolaryngology

President: Dr. W. Howard Morrison, 1500 Medical Arts Bldg., Omaha.
Secretary-Treasurer: Dr. John Peterson, 1307 N. St., Lincoln.

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr. George P. Meyer, 410 Haddon Ave., Camden.
Secretary: Dr. John P. Brennan, 429 Cooper St., Camden.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. Harold J. Joy, 504 State Tower Bldg., Syracuse 2.
Secretary: Dr. Maxwell D. Ryan, 660 Madison Ave., New York 21.

North Carolina Eye, Ear, Nose and Throat Society

President: Dr. Hugh C. Wolfe, 102 N. Elm St., Greensboro.
Secretary: Dr. Vanderbilt F. Couch, 104 W. 4th St., Winston-Salem.

North Dakota Academy of Ophthalmology and Oto-Laryngology

President: Dr. W. L. Diven, City National Bank Bldg., Bismarck.
Secretary-Treasurer: Dr. A. E. Spear, 20 W. Villard, Dickenson.

Oregon Academy of Ophthalmology and Oto-Laryngology

President: Dr. Paul Neely, 1020 S. W. Taylor St., Portland.
Secretary-Treasurer: Dr. Lewis Jordon, 1020 S. W. Taylor St., Portland.
Place: Good Samaritan Hospital, Portland. Time: Third Tuesday of each month.

Pennsylvania Academy of Ophthalmology and Otolaryngology

President: Dr. Lewis T. Buckman, 83 S. Franklin St., Wilkes-Barre.
Secretary Pro Tem: Dr. Paul C. Craig, 232 N. 5th St., Reading.
Time: Last week in April.

Rhode Island Ophthalmological and Otological Society

Acting President: Dr. N. Darrell Harvey, 112 Waterman St., Providence.
Secretary-Treasurer: Dr. Linley C. Happ, 124 Waterman St., Providence.
Place: Rhode Island Medical Society, Library, Providence. Time: 8:30 p. m., second Thursday in October, December, February and April.

South Carolina Society of Ophthalmology and Otolaryngology

President: Dr. J. L. Sanders, 222 N. Main St., Greenville.
Secretary: Dr. J. H. Stokes, 125 W. Cheves St., Florence.

Tennessee Academy of Ophthalmology and Otolaryngology

President: Dr. Wesley Wilkerson, 700 Church St., Nashville.
Secretary-Treasurer: Dr. W. D. Stinson, 124 Physicians and Surgeons Bldg., Memphis.

Texas Ophthalmological and Oto-Laryngological Society

President: Dr. F. H. Rosebrough, 603 Navarro St., San Antonio.
Secretary: Dr. M. K. McCullough, 1717 Pacific Ave., Dallas.

Utah Ophthalmological Society

President: Dr. R. B. Maw, 699 E. South Temple, Salt Lake City.
Secretary-Treasurer: Dr. Charles Ruggeri Jr., 1120 Boston Bldg., Salt Lake City.
Place: University Club, Salt Lake City. Time: 7:00 p. m., third Monday of each month.

Virginia Society of Oto-Laryngology and Ophthalmology

President: Dr. Mortimer H. Williams, 30½ Franklin Rd. S. W., Roanoke.
Secretary-Treasurer: Dr. Meade Edmunds, 34 Franklin St., Petersburg.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. George Traugh, 309 Cleveland Ave., Fairmont.

Secretary: Dr. Welch England, 621½ Market St., Parkersburg.

LOCAL

AKRON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. E. L. Mather, 39 S. Main St., Akron, Ohio.

Secretary-Treasurer: Dr. V. C. Malloy, 2d National Bank Bldg., Akron, Ohio.

Time: First Monday in January, March, May and November.

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. B. M. Cline, 153 Peachtree St. N. E., Atlanta, Ga.

Acting Secretary: Dr. A. V. Hallum, 478 Peachtree St. N. E., Atlanta, Ga.

Place: Grady Hospital. Time: 6:00 p. m., fourth Monday of each month, from October to May.

BALTIMORE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Ernst Bodenheimer, 1212 Eutaw Pl., Baltimore.

Secretary: Dr. Thomas R. O'Rourk, 104 W. Madison St., Baltimore.

Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to March.

BIRMINGHAM EYE, EAR, NOSE AND THROAT CLUB

President: Each member, in alphabetical order.

Secretary: Dr. Luther E. Wilson, 919 Woodward Bldg., Birmingham, Ala.

Place: Tutwiler Hotel. Time: 6:30 p. m., second Tuesday of each month, September to May, inclusive.

BROOKLYN OPHTHALMOLOGICAL SOCIETY

President: Dr. Michael J. Buonaguro, 589 Lorimer St., Brooklyn.

Secretary-Treasurer: Dr. Benjamin C. Rosenthal, 140 New York Ave., Brooklyn 16.

Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

BUFFALO OPHTHALMOLOGIC CLUB

President: Dr. Walter F. King, 519 Delaware Ave., Buffalo.

Secretary-Treasurer: Dr. Sheldon B. Freeman, 196 Linwood Ave., Buffalo.

Time: Second Thursday of each month.

CHATTANOOGA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Each member, in alphabetical order.

Secretary: Dr. Douglas Chamberlain, Chattanooga Bank Bldg., Chattanooga, Tenn.

Place: Mountain City Club. Time: Second Thursday of each month from September to May.

CHICAGO OPHTHALMOLOGICAL SOCIETY

President: Dr. Samuel J. Meyer, 58 E. Washington St., Chicago 2.

Secretary: Dr. W. A. Mann, 30 N. Michigan Ave., Chicago 2.

Place: Continental Hotel, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

CINCINNATI GENERAL HOSPITAL OPHTHALMOLOGY STAFF

Chairman: Dr. D. T. Vail, 441 Vine St., Cincinnati.

Secretary: Dr. A. A. Levin, 441 Vine St., Cincinnati.

Place: Cincinnati General Hospital. Time: 7:45 p. m., third Friday of each month except June, July and August.

CLEVELAND OPHTHALMOLOGICAL CLUB

Chairman: Dr. M. Paul Motto, Rose Bldg., Cleveland.

Secretary: Dr. H. H. Wygand, Guardian Bldg., Cleveland.

Time: Second Tuesday in October, December, February and April.

COLLEGE OF PHYSICIANS, PHILADELPHIA, SECTION ON OPHTHALMOLOGY

Chairman: Dr. W. S. Reese, 1901 Walnut St., Philadelphia.

Clerk: Dr. George F. J. Kelly, 37 S. 20th St., Philadelphia.

Time: Third Thursday of every month from October to April, inclusive.

COLUMBUS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

Chairman: Dr. Erwin W. Troutman, 21 E. State St., Columbus, Ohio.

Secretary-Treasurer: Dr. T. Rees Williams, 380 E. Town St., Columbus 15, Ohio.

Place: University Club. Time: 6:15 p. m., first Monday of each month, from October to May, inclusive.

CORPUS CHRISTI EYE, EAR, NOSE AND THROAT SOCIETY

Chairman: Dr. C. B. Collins, 704 Medical Professional Bldg., Corpus Christi, Texas.

Secretary: Dr. L. W. O. Janssen, 710 Medical Professional Bldg., Corpus Christi, Texas.

Time: 6:30 p. m., third Tuesday of each month from October to May.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Ruby K. Daniel, Medical Arts Bldg., Dallas 1, Texas.

Secretary: Dr. Tom Barr, Medical Arts Bldg., Dallas 1, Texas.

Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. H. C. Schmitz, 604 Locust St., Des Moines, Iowa.

Secretary-Treasurer: Dr. Byron M. Merkel, 604 Locust St., Des Moines, Iowa.

Time: 7:45 p. m., third Monday of every month from September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.

Secretary: Dr. Cecil W. Lepard, 1025 David Whitney Bldg., Detroit 26.

Place: Club rooms of Wayne County Medical Society.

Time: First Wednesday of each month, November to April, inclusive.

Detroit Ophthalmological Society

President: Dr. Raymond S. Goux, 545 David Whitney Bldg., Detroit 26.

Secretary: Dr. Arthur Hale, 1609 Eaton Tower, Detroit 26.

Place: Club rooms of Wayne County Medical Society. Time: 6:30 p. m., third Thursday of each month from November to April, inclusive.

Eastern New York Eye, Ear, Nose and Throat Association

President: Appointed at each meeting.

Secretary-Treasurer: Dr. Joseph L. Holohan, 330 State St., Albany.

Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. Rex Howard, 602 W. 10th St., Fort Worth, Texas.

Secretary-Treasurer: Dr. R. H. Gough, Medical Arts Bldg., Fort Worth, Texas.

Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

Houston Academy of Medicine, Ophthalmological and Oto-Laryngological Section

President: Dr. Lyle J. Logue, 1304 Walker Ave., Houston, Texas.

Secretary: Dr. John T. Stough, 803 Medical Arts Bldg., Houston, Texas.

Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmological and Oto-laryngological Society

President: Dr. Myron Harding, 23 E. Ohio St., Indianapolis.

Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.

Place: Indianapolis Athletic Club. Time: 6:30 p. m., second Thursday of each month from November to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. Edgar Johnson, 906 Grand Ave., Kansas City, Mo.

Secretary: Dr. W. E. Keith, 1103 Grand Ave., Kansas City, Mo.

Time: Third Thursday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

Chairman: Dr. Dennis Smith, 623 Security Bldg., Long Beach 2, Calif.

Secretary-Treasurer: Dr. Robert Null, 710 Security Bldg., Long Beach 2, Calif.

Place: Seaside Hospital. Time: Last Wednesday of each month from October to May.

Los Angeles Society of Ophthalmology and Otolaryngology

President: Dr. Orrie E. Ghrist, 210 N. Central Ave., Glendale, Calif.

Secretary-Treasurer. Dr. K. C. Brandenburg, 110 Pine Ave., Long Beach 2, Calif.

Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:00 p. m., fourth Monday of each month from September to May, inclusive.

Louisville Eye and Ear Society

President: Dr. Joseph S. Heitger, Heyburn Bldg., Louisville, Ky.

Secretary-Treasurer: Dr. J. W. Fish, 321 W. Broadway, Louisville, Ky.

Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

Lower Anthracite Eye, Ear, Nose and Throat Society

Chairman: Each member in alphabetical order.

Secretary: Dr. James J. Monohan, 31 S. Jardin St., Shenandoah, Pa.

Medical Society of the District of Columbia, Section of Ophthalmology and Otolaryngology

Chairman: Dr. P. S. Constantinople, 1835 I St. N. W., Washington.

Secretary: Dr. Frazier Williams, 1801 I St. N. W., Washington.

Place: 1718 M St. N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

Memphis Society of Ophthalmology and Otolaryngology

Chairman: Each member in alphabetical order.

Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis, Tenn.

Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month from September to May.

Milwaukee Oto-Ophthalmic Society

President: Dr. Ralph T. Rank, 238 W. Wisconsin Ave., Milwaukee.

Secretary-Treasurer: Dr. Frank G. Treskow, 411 E. Mason St., Milwaukee 2.

Place: University Club. Time: 6:30 p. m., fourth Tuesday of each month from October to May.

Montgomery County Medical Society

Chairman: Dr. H. V. Dutrow, 1040 Fidelity Medical Bldg., Dayton, Ohio.
Secretary-Treasurer: Dr. Maitland D. Place, 981 Reibold Bldg., Dayton, Ohio.
Place: Van Cleve Hotel. Time: 6:30 p. m., first Tuesday of each month from October to June, inclusive.

Montreal Ophthalmological Society

President: Dr. J. Rosenbaum, 1396 Ste. Catherine St. W., Montreal, Canada.
Secretary: Dr. L. Tessier, 1230 St. Joseph Blvd. E., Montreal, Canada.
Time: Second Thursday of October, December, February and April.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. M. M. Cullom, 700 Church St., Nashville, Tenn.
Secretary: Dr. R. E. Sullivan, 432 Doctors Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to May.

New Haven Ophthalmological Society

President: Dr. William H. Ryder, 185 Church St., New Haven, Conn.
Secretary: Dr. Frederick A. Wiess, 255 Bradley St., New Haven, Conn.

New Orleans Ophthalmological and Oto-laryngological Society

President: Dr. W. B. Clark, 1012 American Bank Bldg., New Orleans.
Secretary: Dr. Mercer G. Lynch, 1018 Maison Blanche Bldg., New Orleans.
Place: Louisiana State University Medical Bldg. Time: 8 p. m., second Tuesday of each month from October to May.

New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. Thomas H. Johnson, 30 W. 59th St., New York.
Secretary: Dr. Wendell L. Hughes, 131 Fulton Ave., Hempstead, N. Y.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

New York Society for Clinical Ophthalmology

President: Dr. Milton Berliner, 57 W. 57th St., New York.
Secretary: Dr. Benjamin Esterman, 983 Park Ave., New York.
Place: New York Academy of Medicine, 2 E. 103d St. Time: 8 p. m., first Monday of each month from October to May, inclusive.

Oklahoma City Academy of Ophthalmology and Otolaryngology

President: Dr. James P. Luton, 117 N. Broadway, Oklahoma City.
Secretary: Dr. Harvey O. Randel, 117 N. Broadway, Oklahoma City.
Place: University Hospital. Time: Second Tuesday of each month from September to May.

Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society

President: Dr. D. D. Stonecypher, Nebraska City, Neb.
Secretary-Treasurer: Dr. W. Howard Morrison, 1500 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m. dinner; 7 p. m. program; third Wednesday of each month from October to May.

Passaic-Bergen Ophthalmological Club

President: Dr. Thomas Sanfacon, 340 Park Ave., Paterson, N. J.
Secretary-Treasurer: Dr. J. Averbach, 435 Clinton Ave., Clinton, N. J.
Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

Philadelphia County Medical Society, Eye Section

President: Dr. Isaac Tassman, 136 S. 16th St., Philadelphia.
Secretary: Dr. Glen Gregory Gibson, 255 S. 17th St., Philadelphia.
Time: First Thursday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. George H. Shuman, 351–5th Ave., Pittsburgh.
Secretary: Dr. Robert J. Billings, 509 Liberty Ave., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

Reading Eye, Ear, Nose and Throat Society

President: Dr. Isaac B. High, 326 N. 5th St., Reading, Pa.
Secretary: Dr. Paul C. Craig, 232 N. 5th St., Reading, Pa.
Place: Wyomissing Club. Time: 6:30 p. m., third Wednesday of each month from September to July.

Richmond Eye, Ear, Nose and Throat Society

President: Dr. Luther C. Brawner, Professional Bldg., Richmond, Va.
Secretary: Dr. Clifford A. Folkes, Professional Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

President: Dr. Frank Barber, 75 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. Charles T. Sullivan, 277 Alexander St., Rochester, N. Y.

St. Louis Ophthalmic Society

President: Dr. C. C. Beisbarth, 3720 Washington Blvd., St. Louis.
Secretary: Dr. H. R. Hildreth, 508 N. Grand Blvd., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting, 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

San Antonio Ophthalmo-Oto-Laryngological Society

President: Dr. Belvin Pritchett, 705 E. Houston St., San Antonio 5, Texas.

Secretary-Treasurer: Lt. Col. John L. Matthews, AAF School of Aviation Medicine, Randolph Field, Texas.

Place: San Antonio, Brooke General Hospital, Randolph Field or San Antonio Aviation Cadet Center. Time: 7 p. m., second Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Roy H. Parkinson, 870 Market St., San Francisco.

Secretary: Dr. A. G. Rawlins, 384 Post St., San Francisco.

Place: Society's Bldg., 2180 Washington St., San Francisco. Time: Fourth Tuesday of every month except June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. David C. Swearingen, Slattery Bldg., Shreveport, La.

Secretary-Treasurer: Dr. Kenneth Jones, Medical Arts Bldg., Shreveport, La.

Place: Shreveport Charity Hospital. Time: 7:30 p. m., first Monday of every month except July, August and September.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. Clarence A. Veasey Sr., 421 W. Riverside Ave., Spokane, Wash.

Secretary: Dr. Clarence A. Veasey Jr., 421 W. Riverside Ave., Spokane, Wash.

Place: Spokane Medical Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. A. H. Rubenstein, 713 E. Genesee St., Syracuse, N. Y.

Secretary-Treasurer: Dr. I. H. Blaisdell, 713 E. Genesee St., Syracuse, N. Y.

Place: University Club. Time: First Tuesday of each month except June, July and August.

Toledo Eye, Ear, Nose and Throat Society

Chairman: Dr. L. C. Ravin, 316 Michigan St., Toledo 2, Ohio.

Secretary: Dr. W. W. Randolph, 1838 Parkwood Ave., Toledo, Ohio.

Place: Toledo Club. Time: Each month except June, July and August.

Toronto Academy of Medicine, Section of Ophthalmology

Chairman: Dr. W. R. F. Luke, 316 Medical Arts Bldg., Toronto, Canada.

Secretary: Dr. W. T. Gratton, 216 Medical Arts Bldg., Toronto, Canada.

Place: Academy of Medicine, 13 Queens Park. Time: First Monday of each month, November to April.

Washington, D. C., Ophthalmological Society

President: Dr. Harold M. Downey, 1740 M St. N. W., Washington, D. C.

Secretary-Treasurer: Dr. Richard W. Wilkinson, 1408 L St. N. W., Washington, D. C.

Place: Medical Society of District of Columbia Bldg., 1718 M St. N. W., Washington, D. C. Time: 7:30 p. m., first Monday in November, January, March and May.

Wilkes-Barre Ophthalmological Society

Chairman: Each member in turn.

Secretary: Dr. Samuel T. Buckman, 70 S. Franklin St., Wilkes-Barre, Pa.

Place: Office of chairman. Time: Last Tuesday of each month from October to May.